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Original Articles

INFECTIOUS (CATARRHAL) JAUNDICE. AN ATTEMPT TO ESTABLISH A CLINICAL ENTITY

Observations on the Excretion and Retention of the Bile Pigments, and on the Blood*

BY CHESTER M. JONES, M.D., BOSTON
AND
GEORGE R. MINOT, M.D., BOSTON

[From the Medical Services of the Massachusetts General Hospital]

INTRODUCTION

The conditions described as catarrhal jaundice, epidemic jaundice, epidemic catarrhal jaundice, infectious jaundice, Weil's disease, spirochetosis ictero-hemorrhagica, spirochetal jaundice, benign infective jaundice, infectious hepatitis, etc., possess many characteristics in common. A survey of the literature reveals a marked state of confusion regarding these conditions and their relations to each other. The purpose of this paper is to attempt to classify more definitely the disease most commonly known as catarrhal jaundice, and to present a series of observations on certain of its characteristic pathological changes.

Typical catarrhal jaundice is sporadic yet it is not uncommon to observe cases develop at the same time in a given group of individuals. This disease resembles in many ways that condition known as epidemic or infectious jaundice not due to spirochetal infection. On the other hand, it resembles only very superficially that specific infectious disease due to the spirochete ictero-hemorrhagica. The latter condition is often referred to as infectious jaundice, thus leading to confusion with cases of infectious jaundice not due to the spirochete. The term Weil's disease has been applied to all types of infectious jaundice and has led to further confusion. It is desirable that this term be abolished from the nomenclature. Examination of standard text-books[1] and monographic articles[2, 3] reveals clearly a lack of uniformity in classifying these various forms of jaundice. It is evident that there are two distinct groups of cases of epidemic jaundice, one due to the spirochete ictero-hemorrhagica, while the etiology of the other is unknown. It is logical, therefore, to classify and describe epidemic jaundice either as spirochetal or as infectious of unknown cause, or if the causative agent is isolated, to add its name. Thus an epidemic could be described as one of infectious jaundice due to

*This paper is No. 3 of a series of articles, a part of the expense of which has been defrayed from a grant from the Proctor Fund of the Harvard Medical School for the Study of Chronic Disease.

B. paratyphosus. It is possible that several distinct micro-organisms may produce an epidemic of jaundice. To the typhoid-paratyphoid organisms have been attributed the cause of some epidemics,[4] while from the initial symptomatology organisms associated with acute infection of the upper respiratory tract have been thought to be the etiological agents in other instances.

Sporadic catarrhal jaundice, from its symptomatology, appears in the nature of an infection, and is spoken of by Brulé[5] as "benign infective jaundice." Clinically these scattered cases differ in no essential way from those observed during the course of at least some epidemics due to unknown cause. Bacteriologically there is as yet little known about either the sporadic or the epidemic type, except in those cases due to the spirochete ictero-hemorrhagica. By analogy, the inclusion of these sporadic cases of so-called catarrhal jaundice in the group of infectious jaundice due to unknown cause is quite comparable to the recognition of interepidemic cases of anterior poliomyelitis, influenza, and meningococcus meningitis. Such a procedure is logical from all clinical considerations, and should lead to a better understanding of the disease in both epidemic and sporadic form. It is therefore proposed to apply the term "infectious jaundice" to cases of both types.

The pathology of infectious jaundice of unknown cause has been inadequately described. Few autopsies have been performed, inasmuch as fatal cases are rare. It has been assumed by many that the jaundice was due to an inflammatory obstruction of the common bile duct, following an ascending infection from the duodenum. The obstruction was popularly thought to consist in a "plug of mucus" in the common bile duct. Little attention was paid to the possibility of liver pathology until the present French school, led by Brulé,[5] Abrami,[6] and others, insisted that the disease involves not particularly the larger bile ducts but either the bile capillaries or the liver parenchyma, or both. Several autopsies reported by Abrami showed no occlusion of any of the larger bile ducts.

CLINICAL DISCUSSION

The presence of jaundice is common to many disease conditions. Hematogenous jaundice is associated *per se* with no characteristic symptoms. Jaundice of hepatic origin, however, as seen in infectious diseases, such as yellow fever and pneumonia, or in cases of mechanical obstruction to the flow of bile, is always attended by a train of very striking and typical symptoms. The character of the latter may be modified or overshadowed by the particular disease in question, but the inherent symptoms of hepatogenous jaundice are largely determined by four distinct factors. These determining features may be stated as follows: (1) The presence of a general infectious process; (2) the existence of a serious disturbance of liver function; (3) the exclusion of bile from the intestine; and (4) the retention of bile in the circulation. Upon the presence or absence of these four factors depends the character of the symptoms to be observed in any jaundice of hepatic origin. In infectious processes such as yellow fever and pneumonia, the last two factors will largely influence the symptomatology, although the essential nature of the disease may predominate the picture. In cases of acute yellow atrophy, diarsenol jaundice, phosphorus poisoning, and the like, a profound alteration of liver function is undoubtedly the underlying cause of the symptoms. Cholemia, always a loose and unsatisfactory term, may also be attributed to the same cause. The gastro-intestinal disturbances associated with mechanical jaundice due to a stone in the common duct or to malignancy are, on the other hand, caused largely by the exclusion of bile from the intestine. Pruritus is probably due entirely to bile retention. The mental depression and bradycardia often seen in hepatogenous jaundice are probably associated with actual liver disturbance.

In so-called "catarrhal jaundice" an understanding of the part played by each of these four factors is essential to an intelligent conception of the disease. In practically every case there is a period, lasting from several days to two or more weeks, which is characterized by symptoms of a type associated with infection and to a disturbance of liver function. Malaise, anorexia, an actual distaste for food, epigastric distress, and a general feeling of wretchedness and mental depression are all present. It is indeed a striking feature of this malady that the severity of the early symptoms is in marked contrast to the paucity of clinical signs. It is extremely important to realize that these symptoms may persist occasionally for two weeks or even longer before the appearance of jaundice. They may be so characteristic as to warrant a tentative diagnosis in spite of the absence of icterus. It is of further importance to realize that clinical jaundice, or jaundice of the skin and sclerae, frequently passes unnoticed if the patient is examined by artificial light. In such cases the finding of bile in the urine is an aid in making a correct diagnosis. Mild cases, especially of the epidemic form, undoubtedly may occur without clinical jaundice or the appearance of bile in the urine, although such cases have an excessive amount of bile in the blood. The absence of clay-colored stools does not preclude the diagnosis of "catarrhal jaundice" inasmuch as in some cases the factor of biliary obstruction is slight.

The duration of the jaundice varies between a few days and several months. The average duration is from two to three weeks. Complica-

tions are rare, and an uneventful recovery is the rule.

It is to be emphasized, therefore, that so-called "catarrhal jaundice" is a malady presenting a characteristic set of symptoms, due essentially to the four factors mentioned above, any one of which may predominate in individual cases. These symptoms are more out of proportion to the clinical signs, and even to the depth of the jaundice, than is usually seen in other forms of hepatic jaundice.

THE CLINICAL MATERIAL STUDIED AND FURTHER CLINICAL DISCUSSION

Detailed observations have been made upon twenty-six cases of infectious jaundice, largely served, but not studied in detail. The clinical findings of all these cases were entirely in accord with the recognized picture of sporadic or epidemic infectious jaundice.

Twenty-three of the cases have been arbitrarily divided into three groups, according to their duration: short, moderately prolonged, and prolonged. In the short cases, twelve in number, the jaundice of the skin and sclerae lasted less than two weeks. In the six moderately prolonged cases it persisted from two weeks to about two months; while in five prolonged cases clinical jaundice was present from sixty to one hundred and ten days. Five patients, whose jaundice persisted for more than four months, will be considered separately from the first three groups. For purposes of comparison,

49.

Case	Duration of Jaundice	Serum Pigment	Duodenal Pigment	Clinical Icterus	R.B.C. in Millions	W.B.C. in Thousands	Differential P.	L.	M.	E.	B.
	Days										
#1	2			++++			60.0	34.0	6.0	0.0	0.0
18♂	4			++++	5.1	5.0	41.0	40.0	18.0	1.0	0.0
	8	45		++	4.9	4.0	46.0	41.0	12.0	1.0	0.0
	12	30		+	4.7	7.0	58.0	35.0	5.0	2.0	0.0
	20	20		tr	4.6	5.1	50.0	38.0	9.0	3.0	0.0
	30	24		tr	4.9	6.0	52.0	26.0	14.0	8.0	0.0
#2	2	54	0	+		5.6	38.0	50.5	9.5	2.0	0.0
21♀	3	81		+		4.0	44.0	42.0	12.0	2.0	0.0
	4	72		+		4.2	47.0	38.0	14.0	1.0	0.0
	5	75		+		4.6	52.0	35.0	12.0	0.0	1.0
	6	90	80	++		3.3	46.0	43.0	7.0	2.0	2.0
	7	85	360	++		3.5	58.5	25.5	13.5	2.0	0.5
	8	80	300	+		5.4	72.0	18.5	8.0	1.5	0.0
	9	40		+		4.0	39.0	42.5	17.5	1.0	0.0
	10	27	500(U)	tr		8.6	55.0	38.0	6.5	0.5	0.0
	11	30		tr		6.8	47.0	42.5	10.5	0.0	0.0
	12	25		0		5.2	46.0	42.5	8.5	2.0	1.0
	13	22		0		5.0	60.0	37.5	10.0	2.5	0.0
	Patient discharged home; exacerbation of jaundice 10 days later, lasting for several days.										
	52	16	96	0		7.2	67.0	24.0	9.0	0.0	0.0
	4			++++		4.0	54.0	40.0	6.0	0.0	0.0
20♀	9			++		3.1	30.0	61.0	8.0	1.0	0.0
	14			+		5.8	35.0	50.0	14.0	1.0	0.0
	21			tr		6.6	40.0	42.0	13.0	5.0	0.0
	30			?tr		5.0	47.0	40.0	10.0	3.0	0.0
	42			0		9.0	52.0	40.0	6.0	2.0	0.0
#4	4			++++		5.0	40.0	48.0	12.0	0.0	0.0
27♀	12			++		5.0	32.0	50.0	16.0	2.0	0.0
	18			+		7.0	52.0	37.0	10.0	1.0	0.0
	40			0		5.8	46.0	41.0	8.0	5.0	0.0
	80			0			59.0	31.0	8.0	2.0	0.0
#5	6			++	5.5	7.2	57.0	36.0	5.0	2.0	0.0
31♀	9			+	4.9	5.0	49.0	41.0	9.0	1.0	0.0
	19			?tr	4.4	6.0	50.0	41.0	4.0	5.0	0.0
	25			0		8.0	54.0	40.0	5.0	1.0	0.0
	39			0	4.8	5.4	57.0	35.0	2.0	6.0	0.0
#6	6	30	56(U)	++	4.7	5.2	40.5	51.5	7.0	1.0	0.0
14♂	7	45				4.6	37.5	54.0	7.0	1.5	0.0
	8		110(U)	+							
	9	32			4.4	5.4	26.5	65.0	7.5	1.0	0.0
	12	36	136	tr	5.1	6.6	44.5	45.5	10.0	0.0	0.0
	17	24	63	tr	3.9	5.6	38.0	47.5	13.0	1.5	0.0
	24	16	42	0	3.6	7.6	53.5	35.0	11.0	0.5	0.0

of the sporadic type, which give further evidence of a pathologic process that involves the entire biliary system. The observations include, in addition to a routine general medical examination, a study of the bile obtained by the duodenal tube, and the bile pigment content of the blood serum. A critical study of the formed elements of the blood has also been made and correlated with the other findings. Four cases were examined at the time of operation, and two cases were studied postmortem. A considerable number of other cases have been observed, but not studied in detail. The clinical findings of all these cases were entirely in accord with the recognized picture of sporadic or epidemic infectious jaundice.

Twenty-three of the cases have been arbitrarily divided into three groups, according to their duration: short, moderately prolonged, and prolonged. In the short cases, twelve in number, the jaundice of the skin and sclerae lasted less than two weeks. In the six moderately prolonged cases it persisted from two weeks to about two months; while in five prolonged cases clinical jaundice was present from sixty to one hundred and ten days. Five patients, whose jaundice persisted for more than four months, will be considered separately from the first three groups. For purposes of comparison, four other cases of liver disease have been added: three of acute yellow atrophy and one of arsphenamine poisoning.

In the above groups of cases the mode of onset was of two distinct types. In about one-half, the initial symptoms were referable to infection of the upper respiratory tract; then followed gastro-intestinal symptoms, and later jaundice. The second form of onset was characterized by the immediate appearance of gastro-intestinal disturbances; jaundice developed subsequently. The two types of onset suggest that different organisms are operative but they

may be merely different manifestations of the same infection.

Early in the disease, practically every case showed, in addition to the jaundice, an enlarged, palpable, and slightly tender liver. Some of the more severe cases presented splenic enlargement. There was no adenopathy. Itching in all types of cases was a frequent and most annoying symptom, and was occasionally the cause of considerable loss of sleep. In every case there was some loss of weight, due largely to a decreased caloric intake and to loss of sleep. In several cases this weight loss amounted in a period of about a month to thirty or more pounds. A moderate elevation of temperature always occurred at the onset. This rise was seldom above 100° F. Fever usually persisted for two or three days. In the most severe cases it was present intermittently, rarely persistently, for several weeks. The course of the disease in all but the very prolonged cases was similar, though there was variation in the intensity and duration of the jaundice. When the jaundice once began to wane, it did so rapidly until only a slight icterus could be detected. The jaundice that then remained disappeared gradually, usually in about two weeks, sometimes in a few days, and occasionally, a trace persisted for a month or more. Convalescence was characterized by these two distinct stages, which are well portrayed in the accompanying charts. Coincident with the rapid fading of the jaundice the symptoms decreased markedly. The liver tenderness disappeared, its size decreased, and the urine and stools approached normal. During the height of the jaundice the urine showed bile, albumin, and casts; in some of the severe cases urobilin and urobilinogen were found. The stools were clay colored before the jaundice lessened and presented the usual characteristics attendant upon the exclusion of bile from the intestines. The urine and stool examinations, however, yielded less information regarding the nature of the disease than the special studies on the bile and blood. In the second stage of convalescence, when the remaining jaundice slowly disappeared, there was no bile in the urine, and there was a rapid return of appetite, strength, and weight. A sense of marked weakness occurred in even the mild cases, and many weeks often passed before full strength was recovered. The twenty-six cases studied in detail were endemic in form though two may well be considered epidemic. Six other cases studied less completely were undoubtedly epidemic in type. It seems that there is no essential difference in the symptomatology

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Case	Duration of Jaundice	Serum Pigment	Duodenal Pigment	Clinical Icterus	R.B.C. in Millions	W.B.C. in Thousands	Differential P.	L.	M.	E.	B.
	Days										
#7	1			++		10.8	48.0	45.0	7.0	0.0	0.0
29 ♂	2			+		5.1	41.0	53.0	6.0	0.0	0.0
	3			+			37.0	53.0	8.0	2.0	0.0
	4			tr		7.6	55.0	35.0	6.0	3.0	1.0
	5			0			54.0	33.5	8.5	3.0	0.5
#8	1	40	50	++	4.4	9.0	48.5	34.5	12.5	3.5	1.0
18 ♂	2	45	59	++	4.2	5.2	47.5	31.5	9.0	8.5	3.5
	4	60	92	+	3.6	9.4	46.5	43.0	3.5	5.5	2.5
	6	27	529(U)	tr	3.4	7.6	44.5	40.0	6.5	7.0	2.0
	8	24	261(U)	?	3.5	7.8	49.0	37.5	4.5	7.5	1.5
	15	19	90(U)	?	4.3	8.0	48.5	39.0	5.0	6.0	1.5
	20	16	61(U)	0	4.0	7.4	51.0	38.0	5.0	4.0	2.0
#9	6	85	108	++	5.7	7.2	55.0	35.0	6.0	3.0	1.0
30 ♂	8		319		5.8	7.2					
	9	64	382	+		6.6	50.0	39.0	8.0	3.0	0.0
	10					6.1	57.0	32.0	7.0	3.0	1.0
	12	32	367(U)	tr		5.8	52.0	32.0	10.5	4.5	1.0
	19	24	168	?		6.6	61.0	30.0	5.0	3.0	1.0
	22	18	160	0		7.0	46.0	47.0	6.0	1.0	1.0
	27	15	109	0	4.6	6.4	57.0	36.0	2.0	5.0	0.0
	38	15	129	0	4.6	5.4	52.0	43.0	3.0	2.0	0.0
	68	10			5.0	6.2					
#10	9	160	114	++	3.9	9.3	59.0	33.0	8.0	0.0	0.0
28 ♂	10		400(U)		4.0	4.6					
	16	60	286(U)			3.2					
	17	60			5.9	5.8	49.0	38.0	9.0	1.0	3.0
	26	20	272(U)	tr	4.8	5.8	53.0	44.0	2.0	1.0	0.0
#11	10			+		12.0	55.5	36.0	5.0	3.5	0.0
44 ♀	13			0		9.6	56.0	52.0	9.5	1.5	1.0
	14			0		10.2	46.0	45.5	4.0	3.5	1.0
	16			0		10.0	43.5	49.0	6.0	1.5	0.0
#12	3			+++		7.0	54.0	34.0	9.0	0.0	3.0
24 ♂	7			+++		5.0	40.0	54.0	5.0	0.0	1.0
	12			++		9.5	67.0	28.0	4.0	0.0	1.0
	17			+		8.8	65.0	21.0	10.0	4.0	0.0
#13	10	108		+++		8.8	69.0	25.0	13.0	2.0	1.0
55 ♂	13					8.6					
	15			++		9.8	48.0	35.0	10.0	6.0	1.0
	19	32		+		16.0	52.0	30.0	10.0	4.0	4.0
	21	32		+			59.0	22.0	13.0	6.0	0.0
	25	28		+			52.0	32.0	14.0	2.0	0.0
	27	28		tr		12.4	57.0	30.0	13.0	0.0	0.0
	31					9.8	63.0	25.0	7.0	4.0	1.0
	33	23		?tr	4.8	10.8	62.0	15.0	18.0	5.0	0.0

of the two types,[7] [8] though it is recognized that in some epidemics the cases may be dangerously ill.[9]

The diagnosis was readily made in most cases when the patient was first seen. In all cases it was established eventually by the course of the disease. Roentgen-ray examination aided to rule out carcinoma and gallstones. No spirochetes were found in the urine or blood of any of the cases.

Treatment consisted in rest, dietary measures, medication, and duodenal lavage. The patients were in bed as long as there was fever or intense jaundice. The diet was comprised of food that could be digested best in the absence of bile in the intestines, namely, food with little fat and high in carbohydrates. The total amount of food, as well as the fat, was increased as the jaundice faded. Medication consisted of the administration of mild sedatives and the external application of anti-pruritics. Ox-bile was given in 15-grain (1.0-gram) doses after each meal as soon as bile was present in reasonable amounts in the intestines, and was continued throughout convalescence. Twenty-one of the patients were treated by duodenal lavage, some but two or three times, several fifteen to twenty times, and one over thirty times. We believe that frequent lavage of the duodenum with magnesium sulphate solution is a valuable therapeutic procedure in hastening recovery.

METHODS

Analysis of the duodenal contents was performed by a modification of Lyon's original technique[10] and has been fully described in a previous article on liver function.[11] Essentially, the method consists in collecting duodenal contents for an hour, following the injection into the duodenum of 60 c.c. of warm 33 per cent. magnesium sulphate solution. Four specimens are collected over consecutive fifteen-minute periods, and are examined spectroscopically. From these examinations the concentration of the bile pigments is estimated. Contrary to the statements found in various articles, it has been found difficult to determine accurately the urobilin content. Bilirubin is normally constantly present in the duodenal contents, and though not giving an absolutely characteristic absorption band, does absorb light from the violet end of the spectrum well into the yellow. If bilirubin is of sufficient concentration this absorption band will merge with that of urobilin and render an accurate reading of the latter impossible. Recently we have devised a method for the separation of these pigments but too late to use it in studying the cases included in this report. In the work here reported the amount of bilirubin and urobilin in the duodenal contents has been estimated together. The characteristic absorption bands of cholecyanin and

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Case	Duration of Jaundice	Serum Pigment	Duodenal Pigment	Clinical Icterus	R.B.C. in Millions	W.B.C. in Thousands	Differential P.	L.	M.	E.	B.
	Days										
#14	25			+		7.0	52.0	26.0	14.0	8.0	0.0
27 ♂	35	30		tr		9.0	50.0	30.0	12.0	8.0	0.0
	41			0		7.0	46.0	37.0	9.0	7.0	1.0
	60	16		0		7.2	54.0	33.0	9.0	4.0	0.0
#15	5			+++	5.3	5.6	68.0	22.0	8.0	1.0	1.0
30 ♂	7	192									
	8	420				4.0	64.0	26.0	7.0	2.0	1.0
	9	430	75	++++		4.0					
	11	750	410(C)	++++		3.2	64.0	29.0	6.0	1.0	0.0
	12	350	243			3.3	67.0	22.0	10.0	1.0	0.0
	13	220	250	+++		4.4	61.0	29.0	4.0	4.0	2.0
	14	320	250	+++		3.9	60.0	29.0	10.0	0.0	1.0
	15	160	230(U)	++	4.8	4.1					
	16		650(U)	++	4.0	4.6	62.0	26.0	9.0	1.0	2.0
	17	160	462(U)	++		4.4					
	18	120	230	+							
	19		211		4.8	5.6	60.0	28.0	8.0	2.0	2.0
	23	84		+							
	25	60	156(U)			9.4	60.0	32.0	8.0	0.0	0.0
	37	54	167(U)	tr		4.2	59.5	28.5	10.0	1.0	1.0
	53	36	151(U)	0	5.7	6.2	58.0	40.0	1.0	1.0	0.0
#18	2	200	0	++	5.7	5.0	51.0	64.0	13.0	1.0	1.0
14 ♀											
	3		0	+++		5.2	31.0	53.0	15.0	0.0	1.0
	4					5.5					
	5	350				6.0					
	6	350	5	++++	5.6	5.4	39.0	55.0	4.0	0.0	2.0
	7		27	++++							
	8	450	31	++++		3.5	44.0	52.0	3.0	0.0	1.0
	9	325	119	++++	5.2	3.8	33.0	61.0	11.0	3.0	2.0
	10		255								
	13	180	205	++	5.2	7.2	58.0	30.0	11.0	1.0	0.0
	15		287	+	4.9	10.4	55.0	34.0	11.0	0.0	0.0
	16		263	tr							
	17	81	250		5.1	5.6	61.0	33.0	5.0	0.0	1.0
	19		218	tr		6.8	45.0	45.0	7.0	2.0	1.0
	21	81	152	tr		6.8	44.0	51.0	3.0	1.0	1.0
	23	108	88								
	28	40	93	?tr	4.9	5.6	52.0	39.0	4.0	3.0	2.0
	31	72		?			52.0	44.0	2.0	2.0	0.0
	38	45	223(U)	?	5.1	7.4	39.0	55.0	4.0	2.0	0.0
	45	40	96	?		7.0	46.0	42.0	8.0	4.0	0.0
	73	27	200	0	5.0	6.4	41.0	50.0	9.0	0.0	0.0
	101	20	157	0	4.7	5.8	40.0	54.0	4.0	1.0	1.0

urobilinogen are not interfered with by the presence of bilirubin, and are easily detected and quantitated. Urobilinogen, except in small traces, is not normally found. Cholecyanin is always an abnormal pigment. An approximate estimation of the concentration of the various pigments in a given specimen is made by taking the actual number of dilutions necessary to obliterate the absorption band of any given pigment. The average pigment value of the four specimens is taken as the final figure for a given collection. A figure of 60 to 100 dilutions has been found to be the usual normal level of the bile pigments (bilirubin and urobilin) in the duodenal contents.

In addition to an estimation of the bile pigments the duodenal sediment was examined microscopically following high speed centrifugalization. Abnormalities in the duodenal sediment have been found to consist of masses of crystals of cholesterin, bilirubin or calcium bilirubin, bile stained leucocytes and epithelial cells, and "casts." The latter consist of cellular débris and bile pigment.

The amount of bile pigment in the blood plasma or serum was taken as an index of the actual degree of jaundice and was estimated by a modification of the method originally described by Blankenhorn.[12] The essentials of the procedure consist in diluting plasma or serum in small test tubes with normal salt solution until no yellow color is visible. The column of diluted serum or plasma is always kept at a depth of 1 cm. The number of dilutions necessary to cause disappearance of the yellow color is taken as a figure for the concentration of bilirubin. Normally the color vanishes before a dilution of 1 to 20 is reached.

The histological observations on the three formed elements of the blood were made by standard technique. Differential counts were made on 200 white cells. Hemoglobin determinations by ordinary colorimetric methods are not accurate in the presence of jaundice, and are misleading. In some cases the oxygen capacity of the blood was determined, and the hemoglobin calculated.

The observations on the duodenal contents

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Case	Duration of Jaundice	Serum Pigment	Duodenal Pigment	Clinical Icterus	R.B.C. in Millions	W.B.C. in Thousands	Differential P.	L.	M.	E.	B.
	Days										
#17	20	210	38	+++	4.8	5.4	60.0	28.0	7.0	2.0	3.0
46♂	21		16								
	23	240	100	+++		5.0	58.0	30.0	10.0	1.0	1.0
	25	190	82	+++		6.0	60.0	29.0	5.0	3.0	3.0
	26		87								
	28	210	70	++		5.8	56.0	35.0	6.0	2.0	1.0
	29		92	++							
	32	130	38			4.8	51.0	40.0	6.0	0.0	3.0
	34		132		4.5	6.8	46.0	42.0	5.0	4.0	1.0
	38	90		++							
	39		135		4.8	5.2	42.0	48.0	4.0	4.0	2.0
	40		105			5.0	50.0	35.0	9.0	4.0	2.0
	42	45	62	+	4.3	6.0	58.0	34.0	5.0	2.0	1.0
	44	60	96	tr	4.3	5.6	41.0	49.0	5.0	4.0	1.0
	49	40	45	tr	4.6	6.6	59.0	34.0	3.0	3.0	1.0
	52	35	46	tr	4.7	6.4	67.0	29.0	3.0	0.0	1.0
	57	30	56	?	4.5	5.2	81.0	17.0	2.0	0.0	0.0
	64	16	80	0	4.8	4.8	62.0	36.0	1.0	0.0	1.0
#18	21	110		++++	4.5	6.2	49.0	40.0	8.0	3.0	0.0
26♂	23		60								
	24	108		+++	4.2	5.8	58.0	27.5	9.0	3.0	2.5
	25		109(U)								
	27		255	+++							
	28				3.2	3.6	57.0	28.5	7.0	6.0	1.5
	29	54	117(U)	++							
	35	90			3.1	8.2	47.5	40.0	6.5	6.0	0.0
	37	60	100	+	3.7	6.4	50.0	39.0	5.5	4.5	1.0
	44	30	107(U)	tr	3.6	8.0	53.0	34.0	4.0	5.5	3.5
#19	21			++++		15.0	85.0	9.0	6.0	0.0	0.0
45♂	22			++++		14.0	67.0	23.0	4.0	6.0	0.0
	23			++++		12.0	59.0	26.0	12.0	3.5	0.5
	26	200	30	++++		13.4	62.0	21.0	14.5	2.0	0.5
	30	200	30	++++		8.5	59.0	30.0	7.0	4.0	0.0
	34					8.9	72.0	19.0	7.0	1.0	1.0
	38	200	77(C)			9.8	59.0	37.0	4.0	0.0	0.0
	39		62	+++							
	41		75			13.5	67.0	24.0	0.0	7.0	1.0
	44	90	87								
	48	60	130			10.4	64.0	24.0	11.0	1.0	0.0
	51	120	97	++++							
	53	110	115	++++	3.6	6.5	60.0	24.0	13.0	3.0	0.0
	55		99		3.9	10.0	56.0	27.0	15.0	1.5	0.5
	59		95	+++			56.0	22.0	17.0	4.0	1.0
	61		107								
	66	80	164	++	4.2	11.0	41.0	54.0	4.0	0.5	0.5
	68		130								
	70	36	70	+	4.5	7.4					
	72	36	77	+	4.5	10.4	59.0	31.5	7.5	1.5	0.5
	74	54	192	tr		6.8	52.0	31.0	7.0	9.0	1.0
	77	40	88	tr		4.4	46.0	40.0	7.0	6.0	2.0
	81	27		?	4.6	8.0	42.0	41.0	6.0	6.0	5.0
	86		98	?	4.6	6.8	51.0	35.0	7.0	1.0	6.0
	88	24	92(U)	0		7.2	44.0	43.0	6.0	4.0	3.0
	102	24	169	tr	5.3	10.6	60.0	24.0	10.0	3.0	3.0
	116	16	165(U)	0	5.2	8.4	58.0	25.0	14.0	2.0	1.0

and blood were made at varying intervals; early in the disease, usually every day or every other day; and later on, at longer intervals. Almost every case was followed throughout convalescence.

STUDIES ON THE DUODENAL CONTENTS

The observations on the duodenal contents were of two types: (a) on the sediment, and (b) on the bile pigments.

The Duodenal Sediment.—The duodenal sediment was of particular interest early in the disease. It contained large numbers of non-bile-stained cells, which were chiefly polynuclear leucocytes, and to a less extent various types of epithelial cells. Mucus was present in abnormal amounts. This was sometimes intimately mixed with bile-stained pus cells and amorphous bilirubin.

The sediment was examined in practically all the cases many times after the disease was well established, and during convalescence. As the jaundice decreased and the bile pigment output began to increase, the sediment consisted of clumps of bile-stained leucocytes, epithelium, masses of precipitated bilirubin, and "casts." The epithelial cells were of two types, columnar and squamous. The latter were small, either circular or roughly cuboidal. At times the columnar cells appeared in a definite fan-shaped arrangement. Casts, appearing similar to those seen in urinary sediments, were frequently observed. They consisted of masses of greenish yellow material, and at times of degenerating epithelial cells, sometimes arranged in acinar fashion. Small plugs composed of cellular débris and inspissated bile appeared in some sediments. Micro-organisms of many types were frequently observed, but no bacteriological studies were made. Rarely, clumps of cholesterin and calcium bilirubin crystals were noted.

This type of sediment just described particularly occurred during the first few days after a flow of bile into the duodenum had been established. The bile-stained cells of all types, as well as the casts and plugs, rapidly disappeared as the jaundice diminished, and but few if any of these elements occurred during the second period of convalescence, when there was only slight jaundice.

The character of the duodenal sediment, and the pus, in particular, indicates that an inflammatory process in the biliary tract is present.

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Case	Duration of Jaundice	Serum Pigment	Duodenal Pigment	Clinical Icterus	R.B.C. in Millions	W.B.C. in Thousands	Differential P.	L.	M.	E.	B.
	Days										
#20	12	180	8	++++	4.2	5.2	65.0	25.0	5.0	5.0	0.0
37 ♀	14	150	26	++++		7.2	60.0	23.0	9.0	6.0	2.0
	17		197(U)	++	3.6	4.4	62.0	28.5	6.0	3.0	0.5
	19	54	282(U)	+							
	21	54	268(U)	tr	3.9	3.6	60.5	29.5	3.0	5.0	2.0
	27	36	172(U)	tr	3.0	6.2	67.0	24.5	6.5	1.5	0.5
	31	28	183	?	3.4	9.4	63.0	29.5	5.0	1.0	1.5
	38	22	180(U)	0	3.9	7.4	64.0	29.0	6.0	0.0	1.0
	45	16	332	0	4.0	6.4					
	47			0	3.8	4.4					
	67	12	140	0	4.4	6.8	60.0	34.0	4.0	2.0	0.0
	103				5.1	10.8	72.0	21.0	4.0	2.0	1.0
#21	8		29	+++							
28 ♂	21		32	+++			59.0	29.0	11.0	0.5	0.5
	23		43	+++		10.1	72.0	18.0	9.0	1.0	0.0
	27		42	+++							
	31		45	+++	4.5	11.2	69.0	16.0	14.0	1.0	0.0
	34		42	++++		6.0	61.0	23.0	13.5	0.5	2.0
	36	700	52	++++		6.3	65.5	23.5	8.5	1.0	1.5
	38		46	++++							
	42	650				6.1	55.0	28.0	13.0	1.6	2.4
	45		110	+++			48.8	32.2	15.4	1.6	2.0
	50	350	142	+++	4.2	5.5	58.0	27.5	9.0	3.5	2.0
	53		125	++							
	57	150	310	++		6.0	59.0	28.5	10.5	1.0	1.0
	60	150	337		4.0	6.9	50.0	40.0	7.0	1.0	2.0
	85	40	393	tr	5.0	4.8	57.0	35.0	6.0	1.0	1.0
	136	18	239(U)	0	5.7	4.8	60.0	34.0	4.0	2.0	0.0
#22	34	200		++	4.7	4.6	67.0	29.0	4.0	0.0	0.0
32 ♀	38		15	++		8.4	61.0	26.0	11.0	2.0	0.0
	41	150		+		7.0	59.0	25.0	13.0	3.0	0.0
	43		25	+++		6.2	75.0	19.0	6.0	0.0	0.0
	45	240	35								
	48		15	+++	5.5	8.2	61.0	31.5	6.5	0.5	0.5
	50	250	35	+++	5.1	5.5	53.0	38.0	6.0	3.0	0.0
	51					8.6	72.5	20.0	7.5	0.0	0.0
	54			+++	4.6	6.2	70.0	24.0	6.0	0.0	0.0
	56				4.9	6.0	64.0	29.0	7.0	0.0	0.0
	61	200	50	+++			62.0	33.5	3.5	0.5	0.5
	62		60	+++		8.0	68.5	24.0	5.0	2.0	0.5
	63		82	++							
	64	144	93	++	4.1	6.2	61.0	24.5	11.0	2.0	1.5
	66		145								
	68	150		++		7.2	62.0	30.0	5.0	1.0	2.0
	70		175								
	72			++	4.1	11.2	65.5	27.0	7.0	0.0	0.5
	73	110	200	+	4.4	13.2	74.0	19.0	5.5	1.5	0.0
	78	54		+	4.5	5.2	57.0	40.0	2.0	1.0	0.0
	92	24	107	?	4.4	6.4	61.0	27.5	10.0	1.0	0.5

It thus contributes evidence of the probable infectious nature of "catarrhal jaundice." The large amounts of non-bile-stained pus observed in some cases at the onset of the jaundice, suggests an initial duodenal infection, although such a finding by no means proves that ascending infection is the constant cause of the process in the biliary tract. The fan-shaped arrangement of the epithelial cells has been noted by Lyon[10] and others in conditions where there is definite biliary tract infection, and such a finding serves as further evidence of such a process in infectious jaundice. It is probable that the "casts" and "plugs" come from the fine biliary radicles. Their presence lends support to the conception that the whole biliary system is involved in this disease, and aids to explain the

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Case	Duration of Jaundice	Serum Pigment	Duodenal Pigment	Clinical Icterus	R.B.C. in Millions	W.B.C. in Thousands	Differential P.	L.	M.	E.	B.
	Days										
#23	21	120		+++		8.4	65.5	27.5	6.0	1.0	0.0
59 ♀	28	108	137	++	4.1	5.6	62.5	28.5	7.0	2.0	0.0
	30	110	145	++	5.0	6.2	70.0	25.5	3.5	1.0	0.0
	32		101	++							
	35	90	107	+	5.2	4.2	69.0	25.0	1.0	3.0	2.0
	38	80	106	+			61.0	30.0	8.0	1.0	0.0
	43	80	116		4.4						
	71	50	111(U)	tr	4.4	7.4	54.0	37.0	8.0	0.0	1.0
	75	48	108(U)	?	4.5	7.2	33.0	61.0	3.0	0.0	3.0
	100	16	85	0	4.7	7.3	48.0	44.0	8.0	0.0	0.0
#24	14	120	8	++		7.8	46.5	37.5	11.5	3.5	1.0
21♂	16	95	12	++		5.4	51.0	41.5	4.0	3.5	0.0
	18		32			3.8	54.5	27.0	13.0	5.0	0.5
	19	125									
	20		60								
	22	72	116	+							
	25		114(U)		4.7	3.9	60.0	30.0	16.5	3.0	0.5
	26	60		++							
	29				4.1	2.8					
	30		6								
	31	150		++		4.0	46.0	39.0	10.0	5.0	0.0
	33	160	6	+++	4.2	3.2	48.0	36.0	13.5	3.5	1.0
	36	195		++++	4.6	6.6	77.5	16.5	3.0	3.0	0.0
	39	120	40	+++	4.9	4.2	55.0	33.5	9.0	2.0	0.5
	41				3.9	5.0					
	42				3.9	5.2	32.5	31.5	9.5	5.5	1.0
	43		87								
	46	70			3.7	4.2	39.0	39.0	17.5	4.5	0.0
	47		17								
	49	162		++++	4.2	5.2	58.0	25.0	11.5	5.0	0.5
	53	280		++++	4.3	6.8	51.5	42.0	4.0	2.0	0.5
	57	150		++++	3.4	4.2	64.0	24.0	9.5	2.5	0.0
	63	170		++++	3.8	2.4	48.5	30.0	15.0	4.5	2.0
	64	Laparotomy; cholecystectomy; liver cirrhosed; common duct thickened; drainage; slow recovery.									
	74						73.5	13.0	10.0	2.0	1.5
	84	180		++++							
	88	72		+++	3.9	4.2	62.5	27.5	6.5	3.5	0.0
	96		73(U)	++	3.0	4.2	55.5	35.0	4.5	4.5	0.5
	102	24	68	+	3.7	6.2	62.5	31.0	4.0	2.0	0.5
	144	15	83	0	4.4	6.3	70.0	22.5	2.5	4.0	1.0
	174	Appetite good; looks pale; sclerae pale; no known jaundice; rather weak; lost some weight. For further details see text.									

nature of the early obstructive process. The study of the bile pigments becomes of particular interest as the abnormal elements of the duodenal sediment disappear, and gives additional evidence that the disease affects the entire biliary tract.

The excretion and retention of the bile pigments were critically studied. The excretion was observed particularly by the output into the duodenum, and the retention by studies on the plasma.

The Excretion of Bile Pigments in the Duodenal Contents.—The duodenal contents contained little or no bile pigment early in the disease. The concentration frequently was found to be from 0 to 12 dilution units. In the mild cases, within a few days the pigment concentration rose to thirty or more units; in the more severe cases the rise took place more slowly, and after a longer period of time. The rise in the concentration of the bile pigments from a very low level to a very high one usually continued rapidly, reaching its peak in a few days. This peak was far above normal in all the cases and in some the pigment concentration was from three to five hundred per cent. of normal. The color of the duodenal contents at such times was a dark mahogany brown, suggesting concentration, but the total flow seemed as copious as usual. At such times it was not uncommon to observe large amounts of urobilinogen, and in two cases cholecyanin was noted. Such findings are distinctly abnormal. As the peak of the pigment curve was reached there was a very rapid clinical improvement. Subsequently, the bile pigments returned to their normal level; and in the mild cases this return took from two to eight days. In the more severe cases there was a distinct delay in the time required for a return to normal, which occupied a period of weeks or even months. In all the cases jaundice of the skin and sclerae disappeared days to even weeks before the bile pigments in the duodenal contents reached the normal level. In several of the mild cases the drop in the bile pigment from the peak was rapid, but the descent stopped at a level well above

the normal and then gradually continued downwards. In the most severe cases not only was the rise of the bile pigments a more gradual affair, but also the fall. In these patients the peak as a rule was not so high as that reached in the less severe forms of the disease.

The Retention of Bile Pigment in the Blood.—The course of this disease can be followed not only by the output of the bile pigments in the duodenal contents but also by the pigment concentration of the blood. Determinations of the bile pigment in the blood permit a more accurate estimation of the degree of jaundice than is possible by simple observation of the patient. The concentration of the bile pigment concentration of bilirubin in the blood reached very high figures. In several instances the serum dilution was over 300, once over 700, and in another very severe case (not described in this series) it was over 1000.

The accompanying charts and tables show the variations in the concentration of the bile pigments in the blood and duodenal contents during the course of the disease. A feature to be noted is that secondary rises in the bile pigment in the blood occasionally happened. These elevations corresponded with changes in the patient's symptoms, appearance, and the excretion of bile into the duodenum.

Examination of the data concerning the bile

56.

Case	Duration of Jaundice	Serum Pigment	Duodenal Pigment	Clinical Icterus	R.B.C. in Millions	W.B.C. in Thousands	Differential P.	L.	M.	E.	B.
	Days										
#25	60	160	15	+++	3.5	11.8	70.5	19.0	9.0	1.0	0.5
19♂	62		30			6.6					
	66		20								
	67	260	10	++++	4.0	3.8	58.5	28.0	9.5	2.5	1.5
	69		10	++++							
	71	210	5	+++	2.9	4.0	56.0	31.0	8.5	3.0	1.5
	73	200			3.5	2.5	61.0	27.0	8.0	3.5	0.5
	76		18	+++		4.0					
	79		18	++++							
	99	250	15	++++	3.3	3.8	34.0	54.0	10.0	2.0	0.0
	102	280	0	++++							
	132	250	25	++++	3.2	3.2	58.0	34.0	7.0	1.0	0.0
	148	140		+++							
	158	128		+++	3.4	3.0	64.0	29.0	3.0	4.0	0.0
	166	250		++++	3.4	2.0	58.0	34.0	5.0	0.0	3.0
	For further details see text.										
#26	60			++++		7.2	73.0	17.0	4.0	6.0	0.0
39♂	63	175				6.2	63.0	26.0	6.0	4.0	0.0
	70	175	b		4.1						
	72	90		+++							
	74	150		++++							
	81	140		++++							
	87	70		+++							
	95	90		+++							
	Patient discharged from hospital feeling much better and less jaundiced; liver condition about the same.										
	18b	180	19		4.4	12.6	79.0	14.0	2.0	4.0	1.0
	Has gained weight; three weeks ago jaundice had practically disappeared but has gradually returned. Anorexia, pruritus.										
	196	324		+++	2.3	13.0	76.5	18.0	3.0	1.0	1.5
	200	300		+++	2.7	7.0	70.5	23.0	3.0	2.5	1.0
	214	100		++	2.9	7.6	74.5	15.0	7.5	2.0	1.0
	249				3.7	9.8	74.0	13.0	10.0	2.0	1.0
	284	64		++							
	302	60	12	++	3.7		83.0	6.0	7.0	4.0	0.0
	312				2.9	8.0	79.0	12.0	4.0	3.0	2.0
	330	250		+++		10.8	74.0	16.0	4.0	3.0	3.0
	340	More jaundiced. During past week had sudden chill, fever, headache, increased weakness, loss of weight and feeling of sickness. Physical examination shows ascites.									
	347	180		++	2.7	15.6	73.0	16.0	6.0	3.0	2.0
	357	64	30-40	+	3.1	9.0	75.0	18.0	7.0	0.0	0.0
	For further details see text.										

of the plasma or serum was essentially a direct reciprocal of the pigment concentration in the duodenal contents. This was most marked early in the disease. Thus with intense jaundice and a high concentration of pigment in the serum there occurred but a small amount of pigment in the duodenal contents. During the period of rapid clinical improvement the bile pigment in the serum showed the greatest drop, but subsequently there was a decided delay before the normal concentration was reached. This delay in certain cases amounted to a period of several weeks after the disappearance of visible icterus, and corresponded to a similar delay in the return to normal of the bile pigments of the duodenal contents. In the severe cases the pigments in the blood and duodenal contents indicates certain facts regarding the pathologic physiology of the disease. Obviously, in the early stages, the jaundice is due essentially to obstruction, because at this time the bile pigments of the blood are high and are absent or low in the duodenal contents. This obstruction is probably due to an inflammatory process, not only in the finer biliary radicles but throughout the biliary tract, and is combined with impairment of liver function. That an inflammatory process is present has already been suggested by the character of the duodenal sediment. As the disease progresses, a rapid subsidence of the obstructive process occurs, as evidenced by the abrupt decrease of the pigment in the blood

and a corresponding increase in the duodenal contents. Following this rapid change it is to be noted that, although the element of obstruction seems to be absent, there is a definite delay in the return of the bile pigments to normal, in both the blood and the duodenal contents. This delay usually covers a period of weeks to months. In spite of the establishment of a pigment output well above normal a high blood bilirubin may persist for weeks, and if of sufficient degree jaundice of the skin and sclerae will be obvious. In this period of the disease the jaundice must be accounted for on another basis than that of obstruction. It does not seem as if abnormal red cell destruction would sufficiently explain this fact, although an increase in blood destruction may be a slight factor in its production. The jaundice in this latter stage of the disease may best be accounted for by injury to the liver cells by some toxic agent associated with the original infection. Such a conception is entirely analogous to that of the cause of the jaundice produced by certain chemical poisons which appear to affect the liver parenchyma. Damage from toxic substances can produce inability of the liver to maintain adequately normal pigment metabolism and lead to excessive pigment excretion. This form of alteration in the liver function has been discussed in a previous article by Jones,[11] and has been commented upon by McMaster and Rous[13] in their work on animals. Evidence that alteration in pigment metabolism exists in infectious jaundice is found not only in the greater concentration of bile pigments in the duodenal contents, but also in the appearance of the abnormal pigment cholecyanin and abnormal amounts of urobilinogen. Further support for the conception that damage to the liver leads to alteration in pigment formation, excretion, and retention, is given by observations made on three cases of acute yellow atrophy and one case each of arsphenamine and trinitrotoluene poisoning. In these patients there was little or no evidence of biliary obstruction, and at times a very large output of bile pigment was associated with marked jaundice. Cholecyanin was present in two of the cases of atrophy. At autopsy all three cases of acute yellow atrophy showed no obstruction to the flow of bile.

In certain cases of infectious jaundice the stools at no time appear light in color. It is, therefore, evident that the obstructive element in this disease may play a minor part in individual cases. It seems logical to conclude, therefore, that in infectious jaundice there exists rather particularly an alteration of the function of the liver due to injury to the liver cells, although the infection may cause sufficient inflammation to permit temporary obstruction involving the biliary tract throughout its many ramifications.

OBSERVATIONS ON THE FORMED ELEMENTS OF THE BLOOD

The literature appears to contain no complete reports on the three formed elements of the blood—the red cells, platelets, and white cells—in sporadic or in epidemic infectious jaundice. We have studied these elements critically, most particularly the white cells, because the change they show from normal is more striking than that of the erythrocytes or platelets.

The typical abnormalities noted below, particularly of the leucocytes, occurred not only in the sporadic cases but were observed in three definitely epidemic cases. This fact perhaps lends support to the view that cases of so-called catarrhal jaundice are but sporadic cases of epidemic jaundice, not due to spirochetal infection. It is of interest to note that similar white cell findings were not observed in a large number of cases of jaundice due to mechanical obstruction.

The Red Corpuscles.—The red corpuscles were not counted in every case. The counts that were made show that in this disease a reduction in the number of red cells is the rule. This reduction seems to be most marked shortly after the height of the jaundice. In the mild cases, the drop in the erythrocytes amounted to even a million per cu. mm. In the more severe cases a still greater decrease was observed, so that in two patients the count was as low as 2,700,000 and 2,200,000 per cu. mm. respectively. In all of the cases with an uncomplicated convalescence the red cells returned to normal slowly over a period of weeks.

The hemoglobin decreased in proportion to the numbers of erythrocytes; relatively, however, somewhat more, so that the color index of less than 1 was the rule. Daily fluctuations of a rather wide degree occurred that suggested that the hemoglobin content in this condition varied with certain alterations in pigment metabolism.

Examination of stained cover-glass preparations showed the red cells to be altered from normal. Slight achromia was usual but never very marked. Slight to moderate variation in size, with still less change in shape, was the rule. Microcytes and larger cells with fine filamentous processes were never plentiful, but a few were present, particularly at the height of the disease. Such cells are associated with blood destruction and suggest that an increase of blood destruction is at least one of the causes of the anemia. The alteration in pigment metabolism is probably a contributing factor, while a defective formation may well play a rôle. That the latter process contributes to the cause of the anemia is particularly suggested by the white cell picture of leucopenia and by the fact that in convalescence evidence of increased blood formation follows a period when active regeneration is not evident. The increased formation

is reflected in the peripheral blood by the appearance of moderate numbers of polychromatophilic cells, by an increase of platelets, and by the number and characters of the white cells yet to be referred to.

The Blood Platelets.—Counts of the blood platelets were not made. From fixed preparations an estimate of their number was recorded. Up to the time of the height of the jaundice they occurred in essentially normal numbers. With decrease of the jaundice they always increased, and even in mild cases frequently rose to a level far above normal. At such times both very small and very large platelets, often abnormally granular, were observed. Such platelets occur with the rapid formation of these elements from their parent cell, the megakaryocyte. In two instances a portion of a nucleus of the parent cell was found.

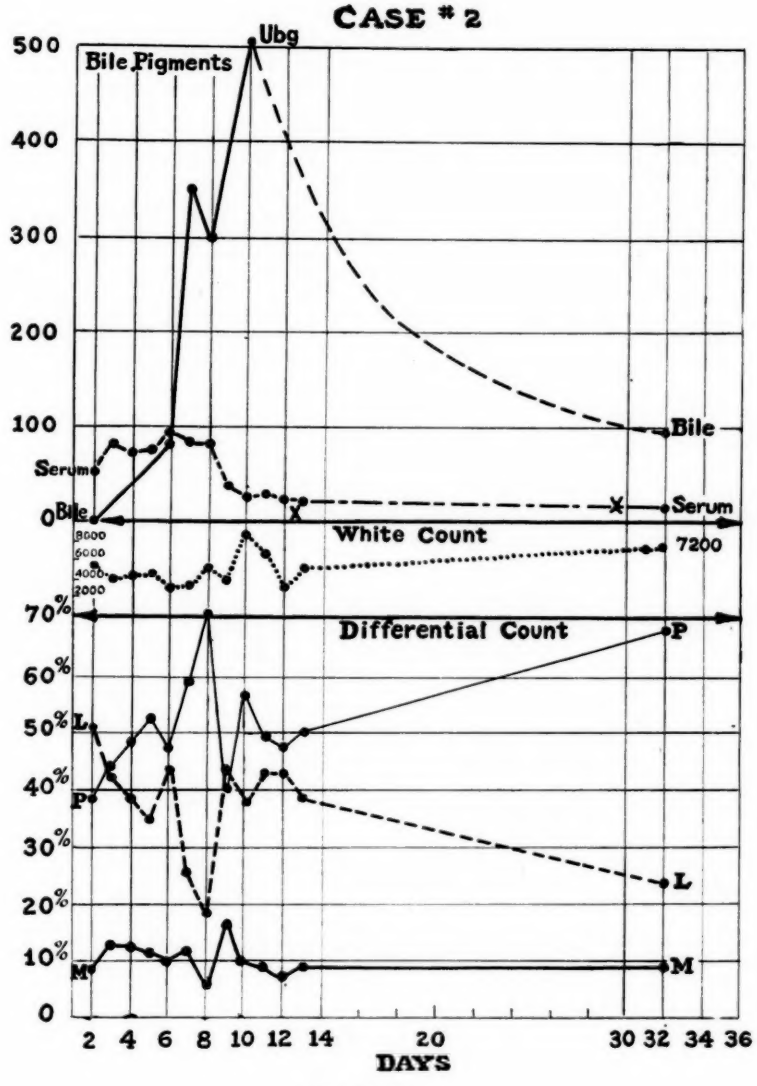

CHART 1

The chart illustrates the usual trend of the white count and the differential count in a typical mild case of infectious jaundice. The course of the jaundice, the excretion and retention of the bile pigments, and the time relations between these processes and changes in the differential count are clearly shown. With the exception of one or two isolated observations the white count was low throughout the disease, and the lymphocytes and large mononuclears were increased. Many of the cells, especially the large mononuclears, showed vacuolization, most marked during the height of the disease. The patient recovered without complications, and the bile pigments, white count, and differential count were normal at the end of a month.

In this and the following charts the same symbols are used. The panel labelled "Bile Pigments" represents the course of bile pigment retention and excretion during the course of the disease. The solid line labelled "Bile" represents the level of bile pigment excretion in the duodenum expressed in dilution units. "Ubg" indicates the presence of abnormal amounts of urobilinogen.

The broken line labelled "Serum" indicates the level of bile pigment in the blood expressed in terms of serum or plasma dilution. Points on the abscissa represent duration of time in days.

Variations in the white count and the differential count in this case are shown in the middle and lower panels. The former is self-evident. Values in the latter are expressed in percentages of the total differential count. P = polynuclear neutrophiles; L = lymphocytes; M = large mononuclears. In this chart all points on a given abscissa are synchronous.

The symbol X in the upper panel ("Bile Pigments") indicates that there was no clinical jaundice.

The White Corpuscles.—Abnormal alterations of the white cells were more striking than the changes in the red cells and platelets. The abnormalities that occurred were referable, not only to the total number and the percentage

number of the various forms, but also to their morphological character.

The total number of cells was usually increased early in the disease. This initial rise in the white cells was seldom above 10,000 per cu. mm.; in one case 16,000 per cu. mm. This leucocytosis lasted for but a few days, rather longer in the more severe than in the milder cases. There then occurred a marked drop in the white cells until in many cases leucopenia was present. The count often went below 4000 cells per cu. mm. The lowest count was 2000 per cu. mm. and occurred in a very severe case. In each case the lowest leucocyte count was usually obtained a few days after the jaundice had started to diminish. The return to a normal level was a gradual one, covering a period of from days to weeks. With the exception of the initial leucocytosis, high white counts were observed only in those cases showing complications.

The quantitative variations of the white cells were quite abnormal, changing with the duration and character of the case. The changes were similar, however, in the cases without complications, so that a rather characteristic differential count occurred. Very early in the disease the differential count was within the normal range though a slight increase of the polynuclear neutrophiles occasionally occurred. Soon after the jaundice began there was a sharp drop in the polynuclear neutrophiles, which was usually coincident with the decrease in the white count and was accompanied by a rise in the lymphocytes and large mononuclears. This rise was nearly always absolute in spite of the leucopenia. The general tendency for an increase of the different forms of mononuclear cells was evident in every case, though, as shown in the preceding tables, there were variations in the individual cases. The lymphocytes were frequently as high as 50 per cent., and occasionally exceeded 60 per cent. In the mild cases, the greatest number of lymphocytes occurred close to the time of the height of the jaundice or shortly thereafter. A tendency to lymphocytosis was evident throughout convalescence. In the more severe cases the lymphocytosis was often less marked and more delayed in appearance than in the mild cases.

The large mononuclears frequently totalled over ten per cent., and the highest percentage recorded was 17, with a white count of 7400 per cu. mm. A sharp distinction between the smaller large mononuclear cells and the larger abnormal lymphocytes is difficult, so that some variations in the figures given for the numbers of these cells may be accounted for by differences of opinion concerning a given cell. Even so, the peak of the large mononuclear count occurred about the same time as that of the lymphocytes. The increase of the large mononuclears usually did not persist for so long a time as that of the lymphocytes. The mononucleosis, like the lymphocytosis, was less marked and more delayed in appearance in the more severe than in the milder cases. During convalescence it was not unusual to find some increase of the large mononuclears, and occasionally a distinct secondary increase in their percentage numbers occurred late in the disease.

As indicated, with the increase of the different mononuclear cells the percentage of polynuclears fell. Thirty-one per cent. of polynuclear neutrophiles with a total white count of 5200 per cu. mm. was the lowest figure obtained.

The polynuclear eosinophiles were, as a rule, within normal limits, though over 5 per cent. was recorded in several cases, and 8.5 per cent. was the maximum. The basophilic polynuclears did not appear in abnormal numbers.

The white cell picture of the very severe cases presented considerable variation, and in contrast to the milder cases showed no tendency to an orderly progressive alteration in the numbers of white cells. The data in the preceding tables show the variations observed in these individuals. Chart 1 shows the fluctuations in the white cells in a typical mild case.

The presence in the peripheral blood of abnormal and immature forms of various types of white cells, shown by their morphological character, was as great a feature in these cases of infectious jaundice as the alterations in the percentage number of white corpuscles. The cells classified as lymphocytes were the ones that showed the greatest variation from normal type. Fifty per cent. of these cells during the period of most marked "lymphocytosis" were often young atypical ones; and at least a few atypical forms were found throughout the period of convalescence. They were observed in varying numbers in every case, being rather more frequent in the milder cases than in the severer ones. These cells resembled somewhat many of those seen in cases of infectious mononucleosis[14] and Snapper, Rijkens and Terwen[15] have reported one such case with the clinical features of infectious jaundice. The atypicality and youth of the cells perhaps conveniently designated as lymphocytes were evidenced by considerable variation in size, and increased amounts of cytoplasm, which was often deeply basophilic, at times contained coarse and abnormal numbers of granules, and often showed elongated protoplasmic processes. The nuclei of these cells were frequently large, pale, and eccentrically placed, so that some cells with very basophilic protoplasm were considered plasma cells.

Immature and atypical forms of large mononuclears appeared with the abnormal lymphocytes, as did occasionally an increased number of broken white cells, so that the picture resembled somewhat that noted by Minot and Smith[16] in tetrachlorethane poisoning. The atypical large mononuclears were characterized by great variation in size, abnormal cytoplasmic granulations and processes, and nuclei which

were usually round, sometimes irregular and unduly vesicular. Speculation concerning the cause of the increase of these cells will be referred to below.

Immaturity of the polynuclear neutrophiles, but not of the eosinophiles or basophiles, was also observed. The youthful forms of these bone marrow elements, with non-lobulated nuclei, varied in size. Their cytoplasm often contained coarse granulations which tended to be either more basophilic or eosinophilic than normal. They were never plentiful and seldom formed more than 5 per cent. of the white cells. They particularly occurred shortly after the jaundice commenced to subside and at the time the platelets were increased, and may be taken to indicate increased marrow activity. Adler[17] has recently described changes in leucocytes that occur in infectious diseases and refers particularly to "toxic" changes seen in the polynuclear series. The plate that illustrates his article shows these cells and also mononuclear elements, many of which resemble the abnormal cells seen in cases of infectious jaundice.

In addition to the morphological changes indicative of youth, the white cells showed another prominent feature, namely, vacuolization. This occurred particularly in the large mononuclears and was usually confined to the cytoplasm of the cells. Rarely, vacuolization of the nucleus was observed. In certain instances nearly all the large mononuclears were vacuolated. Vacuoles were much less common in the polynuclear neutrophiles, and were occasionally present in lymphocytes and eosinophiles. Twelve per cent. of vacuolated cells was a common finding, while as many as 15 to 18 per cent. were noted. Usually single vacuoles occurred, but several in a single cell were not at all infrequent. The vacuoles were clearly defined, round or oval, and ranged in size from a very small point to an area as large as a red blood corpuscle. Vacuolated cells were most frequent as the jaundice started to wane, which was at the time the greatest numbers of immature cells and different mononuclear elements were present. Nevertheless it was not at all uncommon to find such cells towards the end of convalescence even in the absence of visible jaundice.

As convalescence progressed all the white cells gradually became of normal type.

Vacuolization may indicate degeneration but vacuoles occur in actively phagocytic cells. Dieckmann[18] has demonstrated in rabbits inoculated with B. proteus, with resulting liver changes, the occurrence of many vacuolated mononuclear cells in the peripheral blood. These cells were obviously phagocytic, and careful observations seemed to prove that they were particularly derived from the endothelium of the liver and spleen. In the cases of infectious jaundice the cells containing vacuoles were frequently youthful, and except for vacuolization showed no signs indicative of degeneration, thus suggesting that the vacuoles were dependent on phagocytosis. Further support of this view is given by the fact that several times red cells were observed in the vacuoles, and at times basophilic granular material.

It is interesting to speculate on the source of the large mononuclear cells in these cases of infectious jaundice. It seems not unreasonable to believe, as in Dieckmann's animals, that they are particularly derived from the endothelium of the liver and spleen, due to an effect of infection. Quite identical vacuolated mononuclear cells in unusual numbers have been observed by Minot[19] in trinitrotoluol and tetrachlorethane[16] poisoning. It is well recognized that these substances injure the liver. Similar vacuolated cells occur in small numbers in infectious conditions that may produce some effect on endothelium and cause at least mild injury to the liver. Though proof is lacking, we are inclined to believe that in infectious jaundice the large mononuclear cells are especially derived from liver endothelium and that vacuolization is dependent on phagocytic activity.

ILLUSTRATIVE CASES

The five cases presented below in summarized form illustrate the course of the disease in the short, moderately prolonged, and prolonged types of uncomplicated infectious jaundice. Very severe cases with complications will be considered subsequently. The charts for each case show the reciprocal relationship between the retention and excretion of bile pigments. They also serve to show the marked delay in the return of the bile pigments to normal, both in the blood and in the duodenal contents. Tables of the data obtained on the individual cases appear at the end of the article.

SHORT CASES OF INFECTIOUS JAUNDICE

The two following cases illustrate the usual course of a short case of infectious jaundice. Both patients had gastro-intestinal symptoms at the onset of the disease, but otherwise differ in no way from those cases beginning with upper respiratory symptoms.

CASE 8. P. O'N. (W. M. 248635.) Male, age 18.
History: The patient was admitted to the hospital after three days of increasing gastro-intestinal symptoms, particularly nausea and vomiting. There were no other symptoms or significant facts obtained by taking a complete history except that a skin eruption had been present for years.

Physical Examination: The sclerae and skin were slightly icteric. The liver edge was just palpable and was slightly tender. Otherwise the examination was negative except for the presence of eczema.

Laboratory Findings on Admission: Urine: albumin, slight trace; bile, large amount. Stool: light brown in color; soaps, in excess of normal, and free fat present. White count: 7600 cells per cu. mm.

Differential count:		
	Polynuclear neutrophiles	51%
	Polynuclear eosinophiles	1%
	Polynuclear basophiles	1%
	Large mononuclears	11%
	Lymphocytes	36%
		100%

Course: The amounts of bile pigments in the blood and duodenal contents are shown in Chart 2. Following duodenal lavage with magnesium sulphate solution on two successive days the patient felt better, although there was no change in the jaundice. On the fourth day after admission the jaundice began to wane and the gastro-intestinal symptoms subsided. On the twelfth day there was no jaundice of the skin and sclerae.

The highest mouth temperature recorded was 99.5° F. No fever occurred two days after entrance. The white cell count did not go below 5200 per cu. mm. Until discharge, 20 days after admission, the lymphocytes were absolutely increased.

jaundice faded rapidly. The patient was discharged ten days after admission with no symptoms, although he was still slightly jaundiced and the liver was just palpable. Jaundice then disappeared within a few days. While in the hospital there was no fever. The white count was low throughout the disease, with a considerable increase of the lymphocytes and large mononuclears at the peak of the jaundice. Bile pigment estimations are shown in Chart 2.

MODERATELY PROLONGED CASES

The following two cases are examples of the moderately prolonged type of the disease:

CASE 15. J. B. (W. M. 253959.) Male, age 30.

History: The present illness began one week before admission to the hospital with headache, ma-

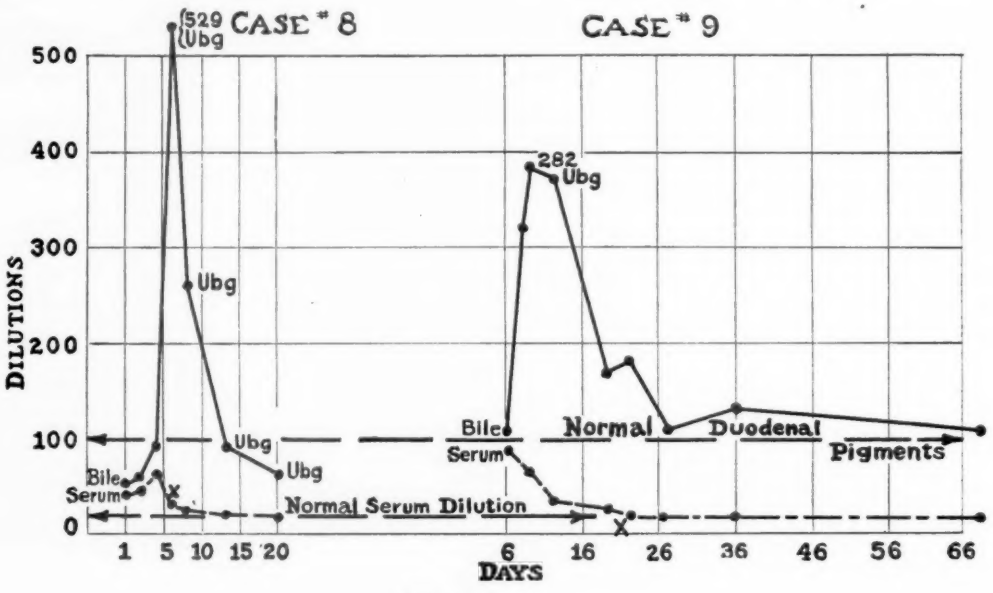

CHART 2

The chart illustrates the course of the disease in two short, rather mild cases of infectious jaundice. The solid line labelled "Bile" indicates the concentration of bile pigments in the duodenal contents. The broken line labelled "Serum" shows the course of the bile pigment in the blood. X = clinical jaundice absent. The sharp rise and fall in the duodenal pigments is striking, as well as their height above normal.

CASE 9. S. P. (E. M. 250112.) Male, age 33.

History: During the four days before admission the patient suffered from dull persistent pain in the back and hips, malaise, headache, and epigastric distress. There were no other gastro-intestinal symptoms. Jaundice had been noticed for two days. The past history was negative.

Physical Examination: Skin and sclerae deeply jaundiced. Liver edge tender and palpable 3 cm. below the costal margin. Otherwise the examination was negative.

Laboratory Findings on Admission: Urine: albumin, slight trace; bile, large amount; sediment contained a few bile-stained casts and epithelial cells. Stool: light brown in color; fat and starch were present. White count: 5800 cells per cu. mm.

Differential count:		
	Polynuclear neutrophiles	62%
	Polynuclear eosinophiles	1%
	Large mononuclears	14%
	Lymphocytes	23%
		100%

Course: Following four duodenal lavages on consecutive days the patient's gastro-intestinal symptoms disappeared, his appetite returned, and the

laise, nausea and vomiting, chills, and slight fever. Gastro-intestinal symptoms remained prominent with marked anorexia. Five days before admission jaundice of the sclerae was noted, and dark urine on the next day. A loss of five pounds of weight occurred during the first week of the disease. The past history was negative except for some unknown type of venereal infection, contracted several years previous.

Physical Examination: This was negative except for the following: the man was deeply jaundiced and had slight epigastric tenderness and spasm. The liver edge was readily palpable and the spleen was felt.

Laboratory Findings on Admission: Urine: albumin, slight trace; bile, large amount; bile-stained cells and casts in the sediment. Stool: light brown in color, with a moderate excess of fat. White count: 5600 cells per cu. mm.

Differential count:		
	Polynuclear neutrophiles	68%
	Polynuclear eosinophiles	1%
	Polynuclear basophiles	1%
	Large mononuclears	8%
	Lymphocytes	22%
		100%

Wassermann reaction of blood serum negative.

Course: The course of this case is shown graphically in Chart 3. In spite of a good output of bile into the duodenum the jaundice increased rapidly for three days, and then as rapidly diminished. During the later period the duodenal pigments were extremely high, and both cholecyanin and urobilinogen appeared. Twenty-five days after the onset of the jaundice the bile pigments of the plasma, though still well above normal, had decreased considerably. Icterus of the sclerae was then still present, and did not disappear until a month later. The bile pigments in the duodenal contents did not return to normal until three months had passed. While in the hospital the patient had no elevation of temperature. The red count fell, particularly in the third and fourth weeks of his illness, dropping from 5,200,000 cells to 4,000,000 per cu. mm. The white count was constantly low. As the disease progressed the lymphocytes and mononuclears increased and remained elevated.

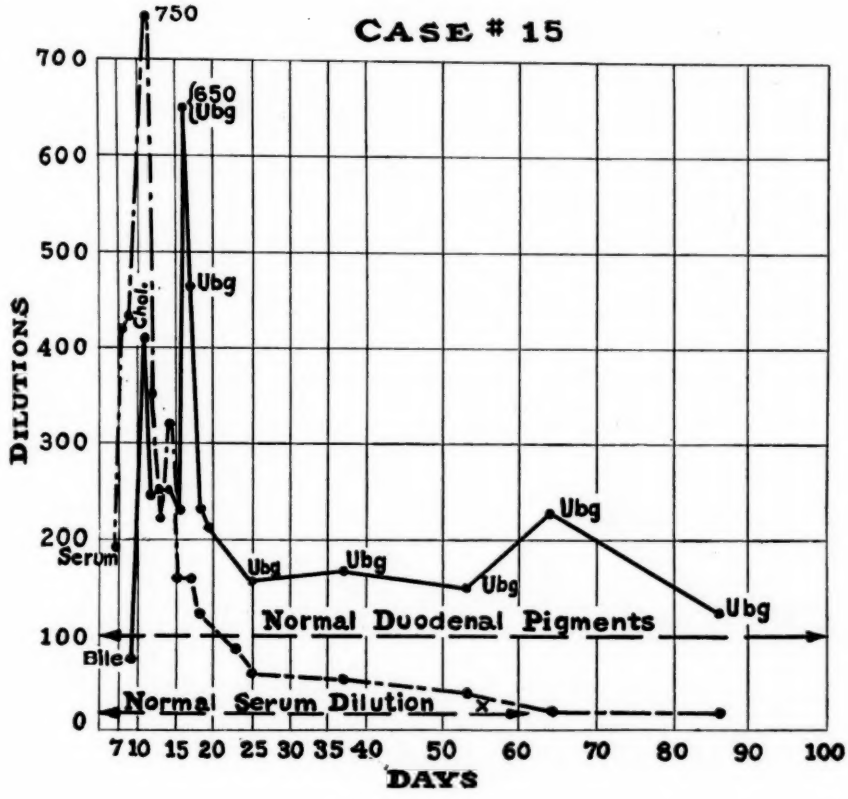

CHART 3

A case of moderately prolonged infectious jaundice. Note the sharp initial rise in serum bilirubin, in spite of a rapidly increasing output of bile into the duodenum, indicating that jaundice associated with obstruction was present for but a few days. The peak of the jaundice is followed by an abrupt drop of the plasma bilirubin to a lower level, with a subsequent drop in the abnormal duodenal pigments. The abbreviation "Chol" seen on the duodenal pigment curve on the 12th day indicates the presence of cholecyanin. The second phase of the jaundice associated with alteration of liver function is evident in this case and the following ones, lasting here from about the 15th to about the 60th day of the disease. It is evident that the jaundice was obstructive in type for only a very few days.

Case 16. H. F. (E. M. 253665.) Female, age 14.

History: The patient felt sick and tired one week before admission. Frontal headache was marked. Two days later the urine became dark and the next day the stools were clay-colored, although jaundice was not noted until entrance to the hospital. For several days nausea, vomiting and abdominal discomfort were marked. Headache, dizziness, and photophobia were the most distressing symptoms on admission. The past history was unimportant.

Physical Examination: The only significant features were moderate icterus, slight epigastric tenderness and a palpable non-tender liver.

Laboratory Findings on Admission: The urine and stool showed the typical findings of obstructive jaundice. White count: 5000 cells per cu. mm.

Differential count:	
Polynuclear neutrophiles	41%
Large mononuclears	15%
Lymphocytes	44%
	100%

Course: The course of the disease in this case is shown in Chart 4 and is quite similar to the preceding one. The jaundice increased for four days, and then diminished rapidly. Slight jaundice remained for a month, when an exacerbation occurred. In the next six weeks the icterus gradually disappeared. On admission to the hospital and for several days thereafter no bile was obtained from the duodenum. Then the flow of bile became established and the bile pigment concentration rose rapidly; after a delay of 10 days it fell rapidly but remained at a high level for about two months. The patient lost 20 pounds in weight in the first four weeks of the disease. About eight weeks after the jaundice started to decrease she had regained her normal weight. The exacerbation of jaundice shown in this case is evidently not unusual, for at least six of the other uncomplicated cases experienced a similar relapse.

On admission the patient's temperature was 100.2° F. It fell to normal in five days and did not rise again. A definite anemia developed during the

disease. Through the entire course of the jaundice the polynuclears were low, and both the lymphocytes and the large mononuclears were markedly increased. Recovery, though much delayed, was uneventful and there were no complications.

PROLONGED CASE OF INFECTIOUS JAUNDICE

The fifth case is an example of a prolonged manifestation of infectious jaundice.

CASE 21. A. T. (P. C.) Male, age 23.

History: For a week prior to the onset of jaundice the patient had progressively increasing symptoms of vague epigastric distress, anorexia, and malaise. The urine then became dark and the stools clay-colored. A temperature of 101° F. was reported four days after the onset of the symptoms, but three days later there was no fever. During the second week, when jaundice was marked, the gastro-intestinal symptoms became milder and the chief complaint was intense itching. The past history was not important.

Physical Examination: The patient was deeply jaundiced and his skin showed many excoriations from scratching. The liver edge was readily palpable and tender. There were no other abnormal signs.

Laboratory Findings: When first seen the urine and stools showed the usual signs of obstruction to the flow of bile. The blood was not examined at this time.

Course: As shown in Chart 5 the course of the disease was a prolonged one, jaundice persisting for about 100 days, and evidences of abnormal pigment metabolism existing for at least 185 days. After the jaundice had been present for about two weeks it became perceptibly deeper and the patient was put to bed. One week later, when the jaundice was still increasing, the serum dilution read 1:700, and only a little bile was entering the duodenum. The patient slept very poorly on account of the severe itching. In the first four weeks of his illness he lost about 35 pounds, and then weighed 150 pounds. Duodenal lavage was started in the third week and was done every three days until the fourth week, when it was performed nearly every day for two weeks. There then occurred a sharp rise in the duodenal pigments, and an equally sharp drop in the plasma bilirubin. The jaundice then diminished rapidly, and coincident with this there was marked improvement in the patient's health. Jaundice of the skin and sclerae gradually disappeared over a period of two months following the abrupt drop in its intensity. The liver remained palpable, however, and slightly tender for 10 weeks.

There was a slight polynuclear leucocytosis in the first weeks of the disease. Leucopenia then developed with an absolute increase in the lymphocytes and large mononuclears. The white cell picture very gradually returned to normal during convalescence. There were no complications, although the duodenal pigments were still abnormally high at the end of six months.

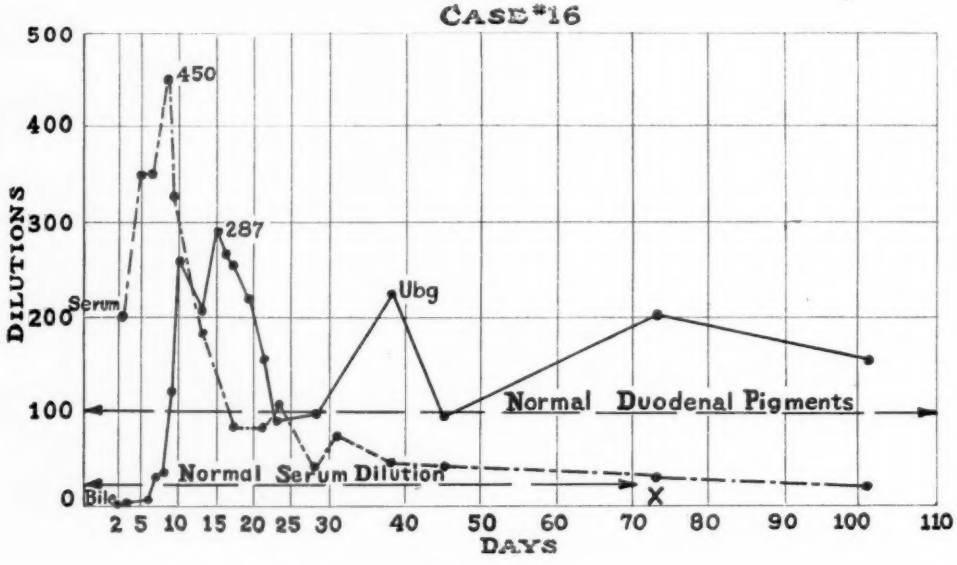

CHART 4

This chart shows the course of a typical case of moderately prolonged infectious jaundice. It is rather similar to the preceding one. The first phase of the jaundice, with obstruction to the flow of bile, is evident early in the disease. The second phase of the jaundice is illustrated clearly. An exacerbation in the intensity of the jaundice is to be noted at about the 30th day, although no decrease in excretion of bile is evident.

VERY SEVERE ATYPICAL CASES AND COMPLICATIONS; A FURTHER CONSIDERATION OF THE RELATIONSHIP BETWEEN EPIDEMIC AND SPORADIC INFECTIOUS JAUNDICE

It is evident that all cases of infectious jaundice, whether mild or severe, present a similar picture, and usually recover without complications. Very severe and atypical cases differ at first but little from the mild ones, but they do so later in that complications develop with serious results. Five very severe and prolonged cases are described below. These, together with a brief consideration of several others, will serve to further illustrate the relationship between epidemic and sporadic infectious jaundice.

Chart 6 illustrates the course of a very severe case. This patient (Case 24) and his brother (Case 25) became jaundiced within a week of

each other. As far as was known they had not come in contact with any one suffering from any recent infectious disease. The general symptomatology and physical findings were similar in each.

CASE 24 (E. M., 247224) was admitted to the hospital two weeks after the onset of jaundice. The onset had been abrupt and was associated with gastro-intestinal disturbances, without pain. Physical examination revealed only a deeply jaundiced young man with a palpable, tender liver. The jaundice was intermittent in intensity (Chart 6) during the following four weeks, and the liver gradually became larger and the spleen palpable. During this time there was no elevation of temperature. The patient's general condition then became gradually poorer, and at the end of six weeks slight tenderness over the gall-bladder was detected, associated with some fever. The temperature gradually dropped from 102° F. to 100° F. during the next few days, but the jaundice increased and the localized tenderness persisted. Operation was decided upon. Prior to operation the red cell count had dropped from 4,700,000 per cu. mm. to 3,400,000 per cu. mm., and the hemoglobin from 110 per cent. (oxygen capacity) to 90 per cent. There was leucopenia during this time, with an absolute increase in the lymphocytes and large mononuclears. After operation the red count fell still lower, so that three weeks later it was 2,560,000 cells per cu. mm. The anemia then gradually lessened, but 14 months after operation the hemoglobin was only 75 per cent. (Tallqvist), although the red count had risen to over 5,000,000 cells per cu. mm. The white cells gradually returned after operation to normal.

At operation the entire biliary tract was found to be involved in an infectious process, the liver appeared cirrhotic and was grossly lobulated by large bands of fibrous tissue. The gall-bladder and larger ducts were markedly thickened. Cholecystectomy with drainage of the common duct was performed. Following the operation the jaundice gradually cleared and has not returned during the past 14 months. During this time the patient has gained weight and strength, but when last seen the liver was still palpable 7 cm. below the costal margin, and the spleen was felt 4 cm. below the ribs. The spleen is larger at present than at the time of operation. Apparently the initial process has been arrested, but the cirrhosis remains.

The second case, Case 25, (W. M. 248828.) Became jaundiced a week after his brother. In spite of the persistence and progressive increase of the icterus, he refused to enter the hospital until two months later, at which time he presented a picture almost identical to that of his brother prior to operative interference. The liver and spleen were both much enlarged. Operation was advised but was refused at this time. After the jaundice had been present for five and a half months, however, subject only to slight variations in intensity, the patient consented to operation. Laparotomy revealed practically the same condition as in his brother, although the liver and spleen were larger, and ascites was present. The gall-bladder was drained. Forty days later the patient died. There was a terminal septicemia. At autopsy the liver was firm, of mottled yellowish brown color, and lobulated. It weighed 1008 gms. Microscopically there was necrosis of the liver cells, with some signs of regeneration and considerable fibrosis. The spleen weighed 453 gms. Microscopically it was not remarkable. Examination of the tissues after silver

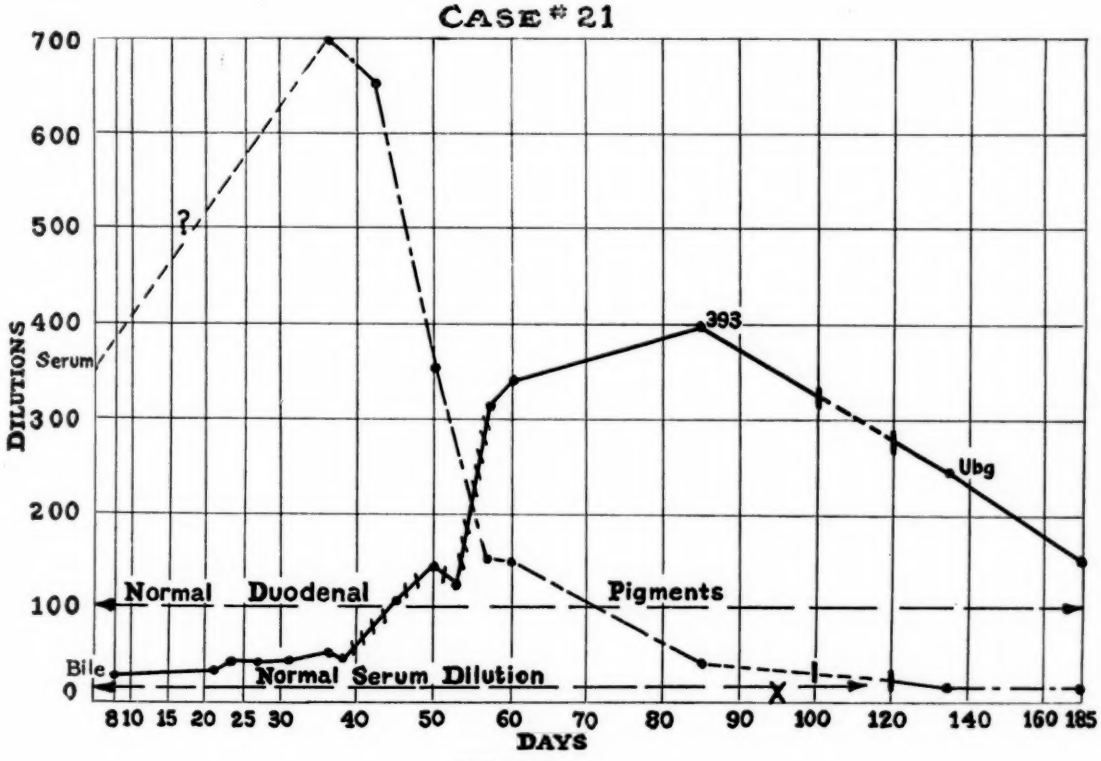

CHART 5

The chart illustrates the course of a serious and prolonged case of uncomplicated infectious jaundice. Note the great depth of the jaundice as indicated by the extremely high serum dilution observed on the 30th day. Also note that on all occasions bile pigments were present in the duodenum. That portion of the curve of bile pigment excretion marked with vertical lines indicates daily duodenal lavage.

impregnation revealed no spirochetes, nor were these organisms found during life in the urine or blood of either patient.

A third very severe case, Case 26, (E. M. 245499.) was followed for about 16 months. The patient, a man of 39 years of age, was first seen after a jaundice of two months' duration. His history was quite similar to that of the two previous cases. The liver and spleen were both palpable and gradually increased in size. For a year he was jaundiced continually, although there were marked variations in the depth of the icterus. At the end of this time operation was advised and performed, because of acute inflammation of the gall-bladder. Laparotomy revealed an inflamed, thickened gall-bladder and a slightly fibrosed liver, with adhesions throughout the biliary tract. There was an abscess between the liver and the gall-bladder. The abscess and the common duct were drained, and for a time the patient improved. The jaundice gradually disappeared, only to return after about three months. The picture was later complicated by tuberculosis, which involved the original drainage wound and caused a persistent sinus. Death finally took place from an acute hemorrhage into the fistula four and one-half months after operation, and 16 months after the onset of the jaundice. At autopsy the liver presented no evidence of tuberculosis. The liver was essentially similar to those described in the two previous cases, although fibrosis was not so marked a feature. In addition to areas of fibrosis of irregular distribution, most marked in the vicinity of the portal veins, examination under the microscope revealed necrosis of the liver cells, and focal aggregations of lymphocytes.

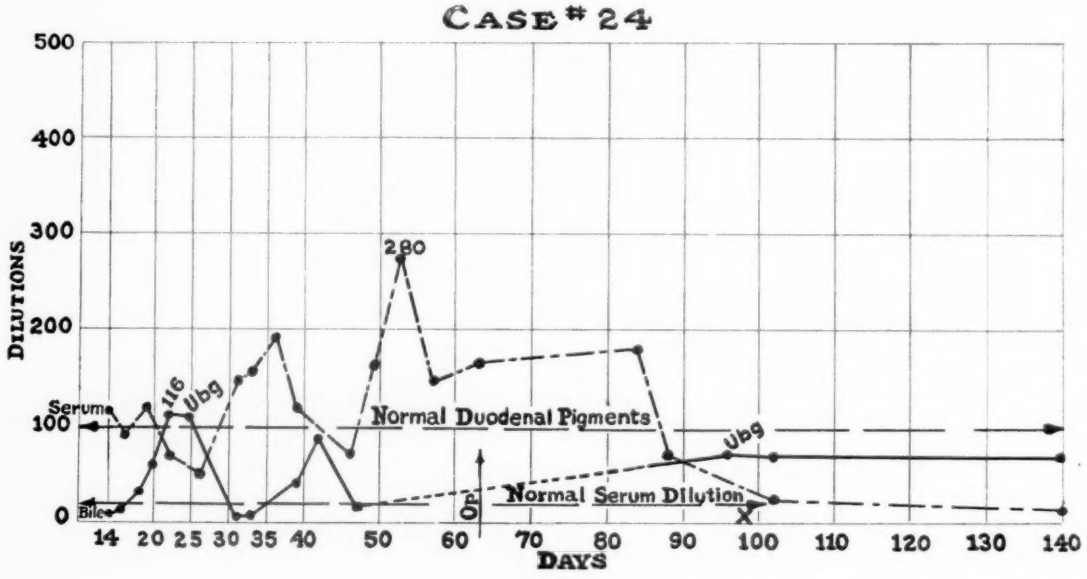

CHART 6

A very severe case (Case 24) of infectious jaundice with complications. The reciprocal relation between bile pigment retention and elimination is clearly shown. For full details consult text.

Other cases that were sporadic in type but otherwise similar to the preceding, have been observed, but only for a limited period of time.

The development of cirrhosis or of hepatitis in cases of undoubted epidemic infectious jaundice is illustrated by the cases referred to below.

The first case is that of a young man of 26 who became jaundiced during an epidemic of infectious jaundice occurring in 1922 in New Haven, Conn.[20] Quite like the other cases of this epidemic, his first symptoms were referable to infection of the upper respiratory tract, with fever and malaise. There then followed gastro-intestinal symptoms, and finally jaundice. Icterus at first increased, then fluctuated in intensity for seven months, during which time loss of strength and weight occurred. At this point in the disease the diagnosis of chronic cholecystitis was made. The gall-bladder was removed and drainage was established by means of a fistula from the common duct. Following operation drainage continued for two months, at the end of which time a second operation was performed, anastomosing the fistula with the duodenum. After that the patient continued to be slightly jaundiced at intervals, and, though not well, his health was better than before operation. Thirteen months after the onset of jaundice, or six months after the first operation, the patient was admitted to this hospital. He showed slight icterus of the sclerae with a high content of bile pigment in the blood. The liver was distinctly enlarged. The spleen could not be felt. There was a moderate degree of anemia with a normal total and differential white count. The ultimate outcome of the case is still in doubt, but it seems probable that permanent improvement is not to be expected.

A second case of similar type was observed in a girl 15 years of age. Jaundice started 14 months prior to admission to the hospital and persisted to date, 16 months since the onset. The early course of her illness was typical of so-called "catarrhal jaundice." Another member of her family, at about the same time, had an apparently typical mild case of infectious jaundice, and recovered completely in three weeks. There were in addition several other similar cases among the students of the school attended by the patient. Unlike the other cases in this epidemic, her jaundice persisted, with variations in intensity. The stools were said to have appeared normal except at the onset of the disease, and during a recent exacerbation which occurred with an upper respiratory infection. She had suffered from progressive loss of strength, weight, and appetite, and from vague gastro-intestinal symptoms. On admission, emaciation and anemia were evident; the liver was slightly enlarged and tender, but the spleen was

not palpable. While in the hospital the patient was drowsy and had marked bradycardia. The duodenal contents on numerous examinations showed a tremendous increase in bile pigment concentration, with large amounts of urobilinogen. Lymphocytosis was present. It is exceedingly probable that this and the previous patient have a diffuse infectious process throughout the liver.

It is obvious from a study of the preceding cases that a serious and even fatal condition may develop in cases of apparently typical epidemic or sporadic infectious jaundice. This condition or complication is in the nature of a cirrhosis or hepatitis. Such a possibility has been mentioned in the literature but does not seem to be generally appreciated.

The white cell picture in these serious cases fluctuated widely, and increases of lymphocytes and large mononuclears were not the rule, as in the milder forms of the disease. The absence of an increase of the different mononuclear elements may perhaps be looked upon as an undesirable sign, while their increase, typical of the less severe cases, may indicate a favorable prognosis.

There is another form of severe liver injury, in addition to that described above, that may rarely develop in cases beginning as typical of sporadic or epidemic infectious jaundice. This condition is known as acute yellow atrophy. Unlike the development of cirrhosis, it usually develops a relatively short time after the onset of jaundice. Cirrhosis of the liver complicating infectious jaundice is essentially a process covering a period of several months. Acute yellow atrophy, on the other hand, is an acute, rapidly progressive condition, although it may not occur until jaundice has been present for several weeks. Such a process may be looked upon as due to the extreme activity of some poison which occurs in all cases of infectious jaundice, but which usually causes little liver damage. In these cases the precise poison is unknown, but the condition is quite comparable to that caused by certain chemical poisons, such as trinitrotoluol. Such a poison commonly causes only a mild toxic hepatitis, but may produce liver degeneration and also atrophy. We have seen at least two cases of epidemic and one of sporadic infectious jaundice which after several weeks of jaundice suddenly became extremely sick and died. In these cases the liver showed the characteristic changes of acute yellow atrophy. It is perhaps significant in this connection to note that in the last three or four years there have been more cases of infectious jaundice and yellow atrophy at the Massachusetts General Hospital than previously. Verbal reports from various physicians also indicate a recent increase in the number of cases of acute yellow atrophy, and it is a recognized fact that in the past few years epidemics of infectious jaundice have been much more frequently reported than in the past.

Serious complications outside of the biliary tract appear to be rare. The term "complication" has been used in the above discussion in reference to certain untoward occurrences observed in the course of apparently typical cases of infectious jaundice. The advisability of such a term may be questioned by some who may believe that the original diagnosis is at fault. We believe, however, that the above cases warrant the use of the term "complication." The first two cases with the infectious cirrhosis occurred in the same family within a week of each other and ran an identical course. The last two cases developed in the midst of well-recognized epidemics, and at the start differed in no way from other cases observed in these epidemics. The final outcome alone differed from the results seen in the other epidemic cases. The serious results reported occurred, we believe, in well authenticated cases of infectious jaundice, were not coincidental, and were probably complications of the original infection.

It is easy to comprehend how cases of undoubted epidemic origin may be considered endemic, inasmuch as the disease contracted in one place may develop weeks later when an individual patient has moved out of the epidemic zone. The recognition of this fact is important in studying the epidemiology of the disease. It is likewise of importance to recognize that the disease may occur in an exceedingly mild form with little jaundice. As already mentioned, it appears that abortive forms of the disease occur with only transient gastro-intestinal symptoms and without clinical jaundice.

The following group of cases illustrates partially the points just referred to. Unfortunately, it was not possible to make complete observations on the patients.

A young woman was seen in Boston, early in April, for acute gastro-intestinal symptoms, consisting of nausea, vomiting, and mild diarrhea. For two days there was a temperature of 101° F. On the fourth day the sclerae were slightly yellow and remained so for five subsequent days, at the end of which time the patient was well. About five weeks previous, her husband had developed a typical case of "catarrhal" jaundice, while in Chicago. He returned to Boston and was at home with his wife about three weeks prior to the onset of her symptoms. His jaundice persisted for about three weeks, and ended with a good recovery. Two days after the wife became sick, her sister, a member of the same household, developed similar symptoms, became jaundiced a week later, and recovered in two weeks. About three weeks after these two women were taken ill, a son and daughter in the family both developed gastro-intestinal symptoms with fever. One recovered in a few days without becoming jaundiced. The other became jaundiced within a week, and remained so for some time. An aunt of these children visited the family at about the time the mother was recovering from her illness. Three and one-half weeks later, after returning to her home in Chicago, the aunt also fell ill with what was described as a typical case of infectious jaundice.

It is not the purpose of this paper to discuss epidemiology further, but from the above account it appears that the incubation period in at least some cases of the disease is about three weeks. In others it is shorter; in the New Haven epidemic it appeared to be about fourteen days. The etiological agent is as yet unknown and, as already suggested, many organisms may perhaps produce a similar picture. The classical endemic cases may well have a quite different etiology from the typical epidemic form, although sporadic cases derived from an epidemic source are known to occur. In most sporadic cases there is no history of contact with individuals who have had the disease. Such contact may, nevertheless, have occurred, or the condition may have been transferred in some other way than by contact between individuals.

SUMMARY

1. Infectious jaundice of unknown etiology occurs sporadically, and is usually known as catarrhal jaundice. It also occurs in epidemic form, and differs in no essential way from the endemic type. The disease usually involves the entire biliary tract and the liver parenchyma by an apparently toxic process comparable to that which occurs as a result of certain chemical poisons.

2. Twenty-six cases of infectious jaundice have been studied critically. Additional observations have been made on a number of other cases.

3. Individual cases vary widely in the severity and duration of the disease, which may persist for a few days or for several months. The usual duration is about four weeks. The intensity of the jaundice varies in degree, and may be very slight or extremely deep. In occasional cases clinical jaundice may be absent. The severity of the early symptoms is in marked contrast to the paucity of clinical signs, and days or weeks may elapse before the development of clinical jaundice.

The symptoms particularly depend upon the presence of a general infection, and a disturbance of hepatic function. The exclusion of bile from the intestine and the retention of bile in the blood are factors of less importance that contribute to the symptomatology.

Recovery from an attack of infectious jaundice is the rule, but a fatal outcome due to untoward complications or sequelae may occur. The latter apparently may consist of a form of liver cirrhosis or of acute liver atrophy.

4. The sediment obtained from the duodenal contents during the early stages of the disease indicates the presence of an inflammatory process in the biliary tract. The duodenal sediment is most characteristic of an infectious process at the time when an increased amount of bile begins to enter the intestine.

5. A study of the retention of bile pigment in the blood and the excretion of the bile pigments into the duodenum shows that during the disease the concentration of the plasma bilirubin is essentially a reciprocal of the bile pigment concentration in the duodenum.

The studies indicate that the jaundice may be separated into two rather distinct phases. Early in the disease there is usually an obstructive phase in which the duodenal contents contain little or no bile. The second phase is marked by an abnormally large output of bile pigment into the intestine. Rapid clinical improvement, with decrease of the jaundice, approximately coincides with the period of greatest elimination of bile pigments into the duodenum. Jaundice of the skin and sclerae may last for months, but disappears days to weeks before the bile pigments reach their normal level in the blood and duodenal contents.

In the cases of short duration there occurs a rapid rise in the bile pigments of the duodenal contents to a very high level, after a brief period of obstruction. This rise may attain a level from 300 to 500 per cent. above the normal. In the more severe and prolonged cases the rise is more gradual, may not be so high, and begins after a longer period of obstruction. Subsequently, in all cases without complications, the bile pigments return to their normal level, at first decreasing rapidly, and then by more gradual stages.

The second phase of jaundice, which is marked by an abnormally large output of bile into the intestine over a relatively long period of time, is apparently associated with an alteration in hepatic function that seems to permit an unusual formation and elimination of pigments. The duration of this second phase of the jaundice is too long to be accounted for by simple retention and subsequent elimination of pigments. On the contrary, it indicates a disturbance of bile pigment metabolism which is of greater importance in the abnormal physiology of the disease and in the continuation of the jaundice than the obstructive phase. The alteration of liver function is evidenced not only by quantitative changes in pigment elimination, but also by the appearance in the bile of the abnormal pigment cholecyanin, and abnormal amounts of urobilinogen.

6. In infectious jaundice typical alterations occur in the blood picture. The three formed elements of the blood, and particularly the white cells, are involved.

A reduction in the number of red blood corpuscles and of the hemoglobin is the rule, and is most marked shortly after the height of the jaundice; a drop of a million red cells is not

unusual, and the red count may fall to about 2,000,000 per cu. mm. Increased blood destruction and altered pigment metabolism probably play a rôle in the production of the anemia, although decreased blood formation seems to be an important feature.

The blood platelets usually increase with a decrease in the intensity of the jaundice.

The total white count may be increased early in the disease. There then occurs a drop in the number of the white cells, until in many cases leucopenia is present. This is most marked a few days after jaundice starts to diminish. The return to normal is gradual, covering a period of days to weeks.

The differential count of the white cells in the initial stage of the disease is often normal, although a polynuclear neutrophile leucocytosis may occur. With decreases in the white count there is an absolute rise in the lymphocytes and large mononuclears. This becomes most marked at the height of the jaundice or shortly thereafter, when the lymphocytes are often 50 per cent. and the mononuclears 12 per cent. of the total count. During convalescence increases in both these types of cells are often observed. In the more prolonged cases the lymphocytosis is less marked and its appearance more delayed. The white cell picture of the most severe cases presents considerable variation, and, in contrast to the milder cases, shows little tendency to an orderly progressive alteration in the numbers of leucocytes.

The presence of immature and abnormal white cells is a feature. The lymphocytes and large mononuclears show the greatest variation from normal, particularly when their numbers are high. Vacuolization of the white cells is commonly observed, and is seen most frequently in the large mononuclears. This feature may be due to phagocytic activity.

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TABLES OF DATA

The preceding tables include the more important data obtained from 26 cases of infectious jaundice. The duration of clinical jaundice in each case is given in days. The figures included in the column headed "Serum Pigment" represent the serum dilution determinations of the bile pigment in the blood. The column "Duodenal Pigment" includes the estimations of bile pigments in the duodenal contents. The letter (U) opposite figures for duodenal pigments indicates the presence of abnormal amounts of urobilinogen. The red cell count is given in millions, and the white cell count in thousands. Under the heading "Differential" the capital letters represent the following:

P=polynuclear neutrophiles.
L=lymphocytes.
M=large mononuclears.
E=polynuclear eosinophiles.
B=polynuclear basophiles.

The numbers of the cases correspond to the numbers already employed in the text. Cases 3, 4 and 5 were of undoubted epidemic origin.

ALCOHOL CHECK EXPERIMENTS WITH PORTABLE RESPIRATION APPARATUS

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APPARATUS constructed on the Regnault-Reiset closed-circuit principle for the measurement of the respiratory exchange of men and of animals has been widely applied and undergone extensive development and much simplification by F. G. Benedict. At the present time the form most commonly employed is the portable respiration apparatus for the determination of oxygen absorption. This is generally used by clinicians in the diagnosis and treatment of pathological conditions in humans.

From the development of the respiration calorimeter at Middletown, Conn., to the present time the most rigid control experiments have been made to determine the accuracy of any type or modification of existing forms in order to insure reliability in observations upon man or animals. The usual method of test has been the combustion of ethyl alcohol in some suitable lamp or burner, with provisions for the accurate measurement of the amount of alcohol burned in a period of time. Ethyl alcohol is particularly suitable for such control experiments. It may be obtained with a high degree of purity, its

purity may be readily determined, and if the alcohol is properly kept, its strength remains unchanged. It has also the very great advantage that in the quantitative relationships of carbon-dioxide production, oxygen use, and heat of combustion, its values are reasonably near to those of man. Thus, by adjusting the quantities used, control values may be obtained of the same order of magnitude as those which may be expected in the application of an apparatus to the measurement of the total metabolism of humans. (See Table 1.)

Table 1. Comparison of gaseous exchange and heat-production per minute of a man under basal conditions with those of absolute ethyl alcohol burning at the rate of 0.165 gram per minute. (Human subject: Body-weight, 70 kg.; height, 170 cm.; age, 30 yrs.)

Conditions.	Carbon dioxide.	Oxygen.	Respiratory quotient.	Heat.
	c.c.	c.c.		cals.
Man	198	241	0.82	1.16
Alcohol	161	241	.67	1.17

The combustion of measured quantities of ethyl alcohol has, therefore, been the standard method of control. This has found application not only in testing the large respiration calorimeter at Middletown, Connecticut,[1] but also the smaller chair calorimeter and bed calorimeter of the Nutrition Laboratory,[2] the clinical chamber respiration apparatus of Benedict and Tompkins,[3] the apparatus for dogs used by Benedict and Homans,[4] and by Benedict and Pratt,[5] and the apparatus employed by Benedict and Talbot in studies with infants and children.[6] Recently the use of a flame of alcohol in an open-circuit current of air has also aided in the standardization of an apparatus for the exact analysis of atmospheric and chamber air.[7] In the development of the first form of breathing-appliance apparatus, viz., the universal respiration apparatus,[8] the use of ethyl alcohol proved impracticable, and the vaporization and combustion of small quantities of ethyl ether was substituted.

In all of these tests the apparatus has been made to conform to the highest degree of accuracy compatible with biological research. Several years ago an extensive comparative study[9] was made of various forms of respiratory-exchange apparatus other than those on the Regnault-Reiset principle, these being specifically the Zuntz-Geppert,[10] the Tissot,[11] and the Douglas[12] methods. In this study the human subject was employed as the control, and the results obtained with each type were compared with those obtained with a standardized apparatus, such as the universal apparatus or the bed respiration calorimeter. With only one of these apparatus (the Tissot or gasometer method) was there any attempt at other methods of control, a few experiments being made with ethyl alcohol in a special form of lamp with moderate success. The results of these comparisons of apparatus justified the belief that the method of control with a human subject was practicable and reliable.

When the portable respiration apparatus[13] was developed, the comparative method of control with a human subject was again used. Two apparatus were employed, the clinical respiration chamber of Benedict and Tompkins,[3] and the Tissot or gasometer method,[11] the latter study being made with 17 medical students.[14] The results of both these series of measurements showed that there was no systematic difference so far as oxygen consumption was concerned, and that the first form of portable apparatus could be used for clinical purposes with an assurance of a high degree of accuracy. When the later form of portable apparatus[15] was devised, *i.e.*, the Benedict and Collins respiration apparatus, it was tested in the same way by means of comparison studies with the earlier form of the portable apparatus. Trained subjects were used and a very satisfactory agreement in results was secured.

In connection with a general program of standardizing various forms of apparatus for studying the respiratory exchange, a series of alcohol check tests was made with the latest forms of portable respiration apparatus. This paper presents the results obtained with the apparatus as regularly used, and also those of several series of experiments with various modifications.

APPARATUS USED IN ALCOHOL CHECK TESTS WITH PORTABLE RESPIRATION APPARATUS

The arrangement of the apparatus used in the alcohol check experiments is shown diagrammatically in Figure 1. The essential features are a lamp, with housing, for burning alcohol, a finely graduated burette for measuring the alcohol, a small hand spirometer for the simulation of breathing, and the type of respiration apparatus to be tested.

Alcohol Lamp.—The alcohol lamp, *A*, is a modification of the one used in the earlier tests with the Tissot or gasometer method.[16] It consists of a housing chamber, *b*, made of glass, and an extension tube, *R*, with a seal, *f*. At the bottom of the housing is a rubber stopper, *g*, through which is inserted a glass tee, *Q*, with a side outlet at *d*. A burner for the alcohol is provided by a capillary Pyrex glass tube, *c*, which is placed in the side outlet, *d*, and held in place by a rubber stopper. A thin piece of asbestos inserted in the enlarged tip of the tube serves as the wick.

Burette and Regulation of Flow.—The method used was devised by one of us[17] and previously employed in the standardization of the infant respiration apparatus. The alcohol supplied to the burner is contained in a burette, *B* (capacity, 10 c.c.), which is graduated to 0.02 c.c., and can be estimated to 0.01 c.c. The graduated portion is 52 cm. in length. The lower part of the burette is connected to the glass tube, *c*, by means of capillary adapters and capillary rubber tubing, *h*. The internal diameter of the tubing is about 1 mm., and the length 85 cm. The rate of flow of the alcohol is controlled by a kymograph, *E*.* The arbor of the kymograph, *H*, is attached to the shaft at the base by means of a metal pin, and a wooden pulley, *G*, 9 cm. in diameter, is fastened to the arbor. Another pulley, *D*, is supported on the stand, *C*. A cord from the wooden pulley, *G*, passes over the pulley, *D*, and is connected with the top of the burette, *B*. The kymograph, *E*, and the stand, *C*, are placed on supports sufficiently elevated above the level of the burner, *c*, to provide for raising the burette, *B*, its entire length. When the kymograph is set in motion, the alcohol flows from the burette into the burner, *c*, the speed of flow being regulated by the size of the fan, *F*, on the kymograph.

Hand Spirometer.—The small hand spirometer, which is made of heavy copper, is the same in shape as that used in a respiration apparatus. Its bell, *J*, is 20 cm. long, and 7.5 cm. in diameter. It is thus possible to get a maximum delivery of 885 c.c. The bell is raised and lowered by means of the handle, *a*, and is immersed in the annular water jacket, *m*. The exit, *p*, is connected to the tubing P P. In use the hand spirometer is moved at a regular rate and height of stroke corresponding to an ordinary human breath with an individual at rest. This is accomplished by means of an electric motor with a reduction gear of such a ratio that the revolutions of a pulley or wheel are approximately 15 to 20 per minute. The transmission from motor to hand spirometer may be made by a cord over a pulley high enough to allow sufficient vertical travel of the spirometer bell. More latitude of adjustment may be obtained by fastening the cord supporting the bell of the spirometer and the cord passing over the pulley from the motor at opposite ends of a walking beam supported on an upright standard. By shifting the points of attachment, the length of stroke of the bell may be varied.

Application of Method to Respiration Apparatus.—This method of testing may be applied to any of the portable respiration apparatus now in regular use, that shown on the right in Figure 1 being the blower type of apparatus, *i.e.*, the form described by Benedict and Collins.[16] Any one of the forms which have two tubes for the ventilation circuit may be tested by this method by detaching the mouthpiece or breathing appliance and connecting the testing apparatus to the ends of the ventilating tubes, as shown in Figure 1.

Course of Air Movement in the Respiration Apparatus.—In the closed-circuit apparatus illustrated in the figure, the air is driven by means of a small blower (a hair-drier), *M*, downwards through a container, *K*, filled with soda lime, then through rubber tubing, *O*, to the bottom of the lamp, upwards over the burner, *c*, through the chamber, *b*, and, in part, through the hand spirometer to the spirometer bell, *N*, of the respiration apparatus. In the forms of apparatus without the hair-drier, the movement of the air is accomplished by the raising and lowering of the hand spirometer, the direction being determined by the placement of valves, such as are used in the Roth modification of the Benedict and Collins apparatus.

PROCEDURE OF AN ALCOHOL CHECK EXPERIMENT

Before beginning the actual determination, the apparatus must first be tested for tightness. For this purpose, all of the parts are connected and the circuit entirely closed, a weight placed on the spirometer bell of the portable respiration apparatus, and an observation made of the height of the bell as read by the pointer on the counterpoise against the millimeter scale. If there is no change after several minutes, the apparatus is considered to be air-tight, and ready for use in the alcohol check tests.

The stopper, *e*, at the bottom and the connection, *f*, at the top of the lamp housing, are first removed. This gives natural ventilation for a current of air to pass through the openings upward through the lamp. The burette is then lowered and both the burette and the rubber tubing filled with alcohol whose composition has previously been accurately determined. In most of the tests here recorded, the alcohol employed had a density of 0.81449 at 20° C. and a percentage composition by weight of 91.288 per cent. Care must, of course, be taken that there are no bubbles of air in the capillary tubing or in the connections between the burette and the lamp. The kymograph is next set in motion; this raises the burette and causes the alcohol to flow into the lamp. A lighted wax taper is passed through the lower opening, and held at the burner tip until the alcohol ignites; the steady flow of alcohol due to the movement of the kymograph keeps the alcohol flame burning. The ventilation is established by an actual draft at first. After the lamp has been burning steadily for several minutes, and it is seen that there is an even flow of alcohol, the lamp is closed by putting in the rubber stoppers at *e* and *f*. Simultaneously, the ventilation in the apparatus is started

*Made by the Harvard Apparatus Company.

by means of an electric current through the hair-drier, and the hand spirometer is set in motion by turning on the current which actuates the connecting motor. The spirometer bell, which lowers rapidly at the time of the starting of the hair-drier, is then immediately filled with oxygen more than sufficient for the expected duration of the period. There is then a continuous oxygen consumption by the alcohol flame and a constant upward and downward movement of the spirometer bell of the respiration apparatus like that which occurs when a human subject breathes into the closed circuit. The apparatus is then ready for the making of the actual observations.

The height of the spirometer bell on the respiration apparatus is first read and simultaneously the height of alcohol in the burette is noted by another individual. The temperature of the air in the spirometer bell is likewise read as soon as possible after the initial spirometer reading. The experiment is then allowed to continue for the length of period planned for and at the end of this time the three readings are again made, *i.e.*, the height of the spirometer bell, the temperature of the air, and the height of the alcohol in the burette. From the difference between the readings of the alcohol burette, one may compute the actual amount of alcohol burned as expressed in cubic centimeters and from this find the amount of oxygen theoretically required to burn the alcohol. Also, from the differences between the spirometer readings and the temperature readings, and taking into account the barometric pressure during the experimental period, one may compute the actual amount of oxygen used from the apparatus in the burning of the alcohol as determined experimentally. The two results may then be compared, *i.e.*, the theoretical amount of oxygen required for the combustion and the actual amount of oxygen used as found. An example is given here of the records made and the calculation of the results.

EXAMPLE OF AN ALCOHOL CHECK EXPERIMENT WITH THE BENEDICT AND COLLINS RESPIRATION APPARATUS

Date: December 20, 1922.

	Alcohol Burette	Height of Bell	Temp. of Bell
	c.c.	mm.	°C.
Beginning	0.30	348	28.0
End	3.80	152	30.5
Difference	3.50	196	2.5
Average temperature			29.3°

Table 2. Results of alcohol check tests with the Benedict and Collins portable respiration apparatus.*

Date.	Duration.*	Alcohol burned.	Rise in temperature.	Oxygen used per minute.*	Per cent of theory.*
	min.	c.c.	°C.	c.c.	%
1922.					
Nov. 3	17.00	3.26	6.9	218	105.0
	12.00	2.00	0.2	182	101.5
	14.00	2.85	1.4	231	105.0
	16.00	2.62	2.0	184	103.5
Dec. 20	10.38	3.74	5.0	409	105.0
	14.31	3.50	2.5	276	104.0
	14.08	4.00	2.4	317	103.0
	21.05	5.40	4.4	291	104.5
	16.88	5.00	4.8	338	105.0
1923					
Jan. 16	(10.00	2.59	6.4	286	102.0
	(10.00	2.53	3.3	287	105.0
	10.00	3.59	2.2	406	104.5
Feb. 9	15.00	2.29	2.9	173	105.0
	15.00	2.43	2.4	180	102.5
	14.00	2.35	3.0	188	103.5
	15.00	2.69	2.8	199	102.0
Mar. 7	10.00	1.88	0.5	215	105.5
	10.00	1.91	0.9	216	104.5
	10.00	1.91	1.7	217	105.5
Average					104.0

*All oxygen figures in this and subsequent tables are expressed to the nearest cubic centimeter, and the percentage figures to the nearest 0.5, except with the averages.
The periods bracketed were made without intermission.

Barometric pressure	777.2 mm.
Duration	14.31 min.
Correction for change of temperature of bell (1.8 mm. x 2.5)	4.5 mm.
Corrected contraction of bell (196+4.5)	200.5 mm.
Calculation of Oxygen Used	*Logarithms*
Change in spirometer bell (200.5 mm.)	2.30211
Factor for bell (21.32 c.c.)	1.32879
Reduction to 0° C. and 760 mm.	9.96525—10
Corrected volume of oxygen used	3.59615
Duration (14.31 min.)	1.15564
Oxygen used per minute	2.44051 or 276 c.c.
Theoretical Oxygen Required	
Burette difference (3.50)	0.54407
Oxygen required per c.c. 91.288% alcohol (1084 c.c.)	3.03503
Total oxygen required	3.57910

Duration (14.31 min.) 1.15564

Oxygen per minute ..... 2.42346 or 265 c.c.
Per cent. found (276 ÷ 265) = 104.2%

RESULTS OF ALCOHOL CHECK TESTS WITH BENEDICT AND COLLINS PORTABLE RESPIRATION APPARATUS

A series of alcohol check experiments was carried out with the Benedict and Collins portable respiration apparatus in which the technique was the same as that generally used in experiments with the apparatus. The results are given in Table 2. In all cases the values obtained for the oxygen used are somewhat higher than theoretical, averaging 4 per cent. above what would be predicted from the amount of alcohol burned.

Table 3. Results of alcohol check tests with the Benedict and Collins portable respiration apparatus, using a weighed cylinder of oxygen.

Date.	Duration.*	Alcohol burned.	Rise in temperature.	Oxygen used per minute.	Per cent of theory.
	min.	c.c.	°C.	c.c.	%
1923					
Jan. 29	9.93	1.75	2.1	194	101.0
	13.37	2.27	1.2	184	100.0
	9.53	1.70	0.7	191	98.5
	11.97	1.94	0.0	172	98.0
	9.78	1.40	0.0	150	97.0
	10.17	1.86	0.7	191	96.5
	11.25	2.09	0.3	200	99.0
Jan. 30	13.17	2.15	1.0	174	98.5
	12.75	2.02	1.2	173	101.0
	9.83	1.56	0.8	172	100.5
	10.92	1.81	0.6	178	99.5
	11.00	1.81	1.2	173	97.0
	9.50	1.71	1.2	201	103.0
	11.75	2.07	0.4	186	97.5
	11.83	2.03	0.5	187	100.5
	12.00	2.00	0.6	181	100.5
	11.17	1.66	2.2	168	104.0
	9.67	1.75	0.9	193	98.5
	10.47	1.96	0.0	199	98.0
	11.42	1.90	0.0	181	100.5
	12.57	2.00	0.8	174	101.0
Average					99.5

* The periods bracketed in this and subsequent tables were made without intermission.

The method of calculating the oxygen used in the combustion of the alcohol is the same as that given by Benedict[13] in his description of the portable respiration apparatus, except for the correction for change in temperature of the air in the spirometer bell during the period. The original correction was an empirical one and was calculated from a series of observations of about 12 minutes' duration by reducing the volume at the beginning of the period to 0°C. and 760 mm. pressure and likewise the volume at the end of the period, and comparing the difference between the two volumes when thus reduced with the quantity calculated from the lowering of the spirometer bell, this also having been reduced. It was found that the volume of oxygen used as thus calculated was greater than that calculated from the contraction of the volume of air in the spirometer as indicated by change in height of the bell. From a number of these calculations an empirical correction *per minute* for each degree rise in temperature of the bell was arrived at and recommended. When the periods are uniformly about 12 minutes in length, this calculated correction can be used. When, however, the periods are of varying length, as in this investigation, the correction should be applied, not on the *per minute* basis but on the whole contraction before reduction to 0°C. and 760 mm. pressure, as the change due to a 5° rise in temperature is the same whether it takes place in 8 minutes or in 15 minutes. In these experiments a new correction has been calculated which is 1.8 mm. (or 38 c.c.) per degree (Centigrade) rise in temperature. This correction has been applied to all the experiments with the Benedict and Collins portable apparatus. The new method of correction makes only a very slight difference in the final results, and plays no significant rôle in the 4 per cent. error.

POSSIBLE FACTORS CAUSING DIFFERENCE BETWEEN DETERMINED AND THEORETICAL VALUES

In view of the fact that the results obtained averaged 4 per cent. higher than the theoretical values, a systematic study was begun to find the cause for the difference. Theoretically, a slight correction should be made for the presence of carbon dioxide in the spirometer bell. When calculated, this would amount to 0.4

or 0.5 per cent.; consequently, when the contraction of the volume of air in the bell took place, there was not only the contraction due to the use of oxygen but also that due to the condensation of carbon dioxide in its absorption by the soda lime.

In one of the first comparisons made, an oxygen cylinder was used to measure the oxygen absorption rather than the contraction in volume from the change in the height of the spirometer bell. By this method of measurement, the apparatus was kept at a constant point so that one did not have the possibility of change due to change in volume. The results are given in Table 3. In these data, the values are slightly lower than theoretical but closer than was found with the usual technique as shown in Table 2, and within the limits of error one might expect with an apparatus of this type.

To substantiate this difference between the measurements made with an oxygen cylinder and those determined from the change in height of the spirometer bell, a series of periods was carried out with a human subject who was extraordinarily well trained and who was accustomed to the portable apparatus, having served as subject frequently. The comparisons were made by using in the first period the loss in weight of the cylinder of oxygen as the basis for calculating the oxygen absorbed, and in the succeeding period the oxygen consumption was calculated in the regular way from the difference in height of the spirometer. Thereafter the methods alternated until eight successive periods had been carried out. The results are given in Table 4.

Table 4. Results of a comparison of measurements of oxygen absorption from contraction of volume of air in spirometer and from loss in weight of an oxygen cylinder with a human subject and the Benedict and Collins portable apparatus.

Time.	Duration.	Rise in temperature.	Oxygen measured by:	
			Cylinder.	Spirometer.
a.m.	min.	°C.	c.c.	c.c.
9.45	10.62	2.2	193	---
10.15	10.00	1.2	---	201
10.35	10.14	0.9	193	---
10.55	10.00	0.8	---	199
11.20	10.10	0.9	189	---
11.45	10.00	1.3	---	197
p.m.				
12.10	9.25	1.0	189	---
12.35	10.00	1.2	---	197
Average			191	199

Three observers checked all readings, and in the periods when the regular method of calculation was used, readings were taken of the height of the spirometer bell every minute at the end of a normal expiration. In the periods with the oxygen cylinder the oxygen consumption ranged from 189 to 193 c.c., while in those in which the change in height of the spirometer was used, the values ranged from 197 to 201 c.c. per minute. The highest value with the cylinder measurement was thus lower than the lowest with the spirometer measurements. The values in the two groups agree remarkably well within themselves, the range in both cases being 4 c.c. In each group the last two periods give identical values. The average value for the cylinder measurements is 191 c.c. and with the spirometer measurements 199 c.c. There is thus a systematic difference of 4 per cent. between the values obtained by the two methods of measurement.

On the supposition that moisture may be a factor in causing the error, a study was made of the moisture conditions inside the spirometer bell, first with burning alcohol and later with a human subject. The method of study was similar to the one used by Roth in his observations of the moisture conditions inside the Roth modification of the portable apparatus.[18] A rubber tube was conducted through an opening in a tee at the bottom of the apparatus and passed up inside the chamber holding the soda-lime can so that its exit lay at a point in the spirometer bell above the hair-drier. The tube led out to two U-tubes containing pumice stone and sulphuric acid, then through a meter, next through another tube containing sulphuric acid and pumice stone, and finally through a hand bulb with two valves, and back into the apparatus at another point in the circuit.

During the alcohol check experiment air was pumped by hand through the U-tubes and meter and back to the apparatus. Thus, samples of air from the interior of the bell were passed through the U-tubes and measured. The beginning weight of the U-tubes was recorded, together with the temperature of the meter. The gains in weight of the tubes after 10 liters of air had been passed through ranged from 0.12 to 0.13 gram and calculations showed that this amounted to about 12 mm. pressure.

A human subject was then substituted and two measurements carried out in the same way as with the alcohol. These likewise gave increases of 0.12 and 0.13 gram for 10 liters of air, with about the same amount of pressure as in the alcohol check experiments. A confirmation of these results was obtained by using a wet and dry bulb psychrometer placed in the circuit immediately preceding the entrance of the air into the spirometer bell. The difference between the readings of the wet and dry bulbs, both in an alcohol check experiment and with a human subject, showed essentially the same tension of aqueous vapor as indicated by the gain in weight of the U-tubes. Thus we have two factors which could account for the discrepancy between the values actually determined and the theoretical values, namely, the presence of 0.4 to 0.5 per cent. of carbon dioxide in the spirometer bell, and the variation of 1.5 to 2.0 per cent. due to pressure of aqueous vapor.

Another factor which undoubtedly contributed to the prevention of complete agreement is the difficulty in obtaining a true value for the average temperature of the whole apparatus. It must be recalled that the calculation of the oxygen used presupposes that the temperature of the air in the spirometer bell at the beginning and end of the period of measurement represents the average temperature of the apparatus as well as the changes in temperature. It has always been supposed that the rise in temperature of the air in the bell was caused by the heat generated by the blower, but blank experiments in which no carbon dioxide was absorbed by the soda lime, and the only probable source of heat was the action of the blower, showed that the rise in temperature due to this factor is very small. Examination of the soda-lime container after successive experimental periods showed that it had become much heated, so it is reasonable to suppose that the air immediately surrounding the container was warmer than the air in the spirometer bell, although part of the air coming into the apparatus undoubtedly passed around the soda-lime container. Accordingly, it would be theoretically possible for the average temperature of the air in the apparatus to be slightly higher than that recorded as the temperature of the air in the bell, which would lead to an error in the direction indicated by the results, namely, the amount of oxygen calculated would then be greater than the actual amount of oxygen absorbed. Measurements of the temperature of the air leaving the soda-lime container showed that after a period or two, the actual temperature of this air was higher than the temperature registered in the bell.

ALCOHOL CHECK TESTS WITH WILSON SODA LIME

Wilson soda lime[19] has a much higher moisture content than the soda lime generally employed. It was thought that if this moist reagent were used and the results were calculated to 0°C. and 760 mm. pressure on the basis that the air in the spirometer bell was saturated, better results would be obtained. While it is considered that with this soda lime the air is actually but 80 per cent. saturated, the extra reduction on the basis of 100 per cent. could be regarded as correcting for the carbon-dioxide content of the spirometer air. A series of tests was made with the Benedict and Collins portable apparatus by the usual method in which the container was filled with Wilson soda lime. The results are given in Table 5. The amount of alcohol burned varied from 1.63 to 2.95 c.c. and the oxygen used from 113 to 216 c.c. per minute. The oxygen found as compared to the theoretical requirement varied from 99 to 102.5 per cent. The average of 14 periods was 100.3 per cent. It is thus seen that, with the Wilson soda lime and with the calculation based on the assumption that the air was completely saturated, the results had a high degree of accuracy.

Table 5. Results of alcohol check tests of Benedict and Collins respiration apparatus, with Wilson soda lime.

Date.	Duration.	Alcohol burned.	Rise in temperature.	Oxygen used per minute.	Per cent of theory.
	min.	c.c.	°C.	c.c.	%
1923					
April 14	15	2.85	2.1	210	102.0
	15	2.95	1.7	216	100.5
	15	2.83	2.0	205	100.5
	15	2.77	2.0	200	100.0
	15	2.31	1.5	171	102.5
	15	2.82	1.5	204	100.0
April 21	15	2.74	1.6	198	99.0
	15	2.83	2.1	204	99.5
	15	2.63	1.4	189	99.5
	15	2.51	1.7	181	100.0
	15	2.50	1.6	181	100.5
	16	1.68	0.9	113	99.5
	15	1.63	0.5	117	99.5
	15	2.32	0.8	162	101.0
Average					100.3

ALCOHOL CHECK TESTS WITH A PORTABLE APPARATUS HAVING AN OUTSIDE BLOWER

The earlier form of the Benedict portable respiration apparatus has been made commercially and is now on the market in a modified form in which a rotary blower outside of the spirometer is substituted for the air impeller inside. The soda-lime container is made in the shape of a cup holding about a quart of the reagent. This cup fits into an opening inside of the apparatus. The direction of the ventilation is the same as with the Benedict and Collins apparatus.

A series of alcohol checks was conducted with this commercial apparatus* in which both the

*We are indebted to Dr. Frank H. Lahey for the opportunity to work with this apparatus (Sanborn, maker).

regular soda lime and the Wilson soda lime were used. The results are given in Table 6. In the first five periods the ordinary soda lime was employed, and when the oxygen consumption is calculated on the dry basis, the average is 103 per cent. The last four periods on June 30, and those on July 2, were with the Wilson soda lime and the results, when calculated on the basis of complete saturation, average 99.5 per cent. of the theoretical.

In addition to the series of observations recorded in Table 6, a series of 13 periods was carried out with this apparatus, in which a reagent was used which is sold under a trade name by the manufacturer of the modified apparatus. The reagent is manufactured under the same patent number as the Wilson soda lime, and apparently has about the same amount of moisture. When the calculations are made on the basis of saturation, the results average about 100 per cent. of theoretical, with a range from 99 to 101 per cent.

Table 6. Alcohol check tests with a portable respiration apparatus with blower outside of spirometer.

Date.	Duration.	Alcohol burned.	Rise in temperature.	Oxygen used per minute.	Per cent of theory.
	min.	c.c.	°C.	c.c.	%
1923					
June 30	12	2.32	1.7	216	103.0
	12	2.42	1.3	225	103.5
	12	2.29	0.9	212	102.5
	12	2.15	0.5	204	105.0
	12	2.28	1.3	210	102.0
Average					103.0
June 30	12	1.85	2.0	164	98.5*
	12	2.13	0.9	188	97.5*
	12	2.18	0.8	194	99.0*
	12	2.01	0.3	182	100.5*
July 2	12	2.41	2.4	220	101.0*
	12	2.28	1.2	205	99.5*
	12	2.43	1.7	223	101.5*
	12	2.13	0.8	195	101.0*
	12	2.13	0.4	191	99.0*
	12	2.22	0.7	197	98.5*
Average					99.5*

*Wilson soda lime was used, and calculations were made on the basis of complete saturation.

ALCOHOL CHECK TESTS WITH THE ROTH MODIFICATION OF THE BENEDICT AND COLLINS PORTABLE RESPIRATION APPARATUS

A series of alcohol check experiments was likewise made with the Roth[20] modification of the Benedict and Collins portable respiration apparatus. The results are given in Table 7. The amount of alcohol burned varied from 1.43 to 3.16 c.c., and the oxygen found varied from 120 to 279 c.c. per minute. The determined values for the oxygen were thus from 98.5 to 103 per cent. of the theoretical values, with an average of 100.2 per cent. The regular soda lime was used in the tests of January 17 and the Wilson soda lime in the tests in April. As recommended by Roth, in calculating the results of the April experiments, a 2 per cent. correction was applied. It will be seen from the table that this type of apparatus gives very satisfactory results. The course of the air current is the reverse of that in the Benedict and Collins type as the air passes from the subject to the soda lime and from there to the spirometer bell. The air in the bell is thus carbon-dioxide free and any heating effect caused by its passage through the lime is recorded directly on the thermometer in the bell. The very low saturation occurring when the regular soda lime is used appears to have no effect upon the results, this being neglected in the calculations. The change in moisture conditions in the bell, due to the Wilson soda lime, is apparently taken care of by the 2 per cent. correction. The correction used for change in temperature of the spirometer was that recommended by Roth, viz., 0.5 mm. per degree Centigrade rise.

ALCOHOL CHECK TESTS WITH THE BENEDICT STUDENT RESPIRATION APPARATUS

One of the earliest groups of alcohol check tests made with the arrangement described in this article was carried out with the student respiration apparatus devised by Benedict and Benedict.[21] Subsequently other tests were made with the apparatus.

In this simplified form of respiration apparatus the air in its passage to and from the subject passes through valves into and out of a metal can, partially filled with soda lime. Provision for expansion and contraction of the volume of air is made by a rubber diaphragm attached to the top of the can. Known amounts

of dry air are forced into the soda-lime can through a 3-way valve at such intervals as will keep the diaphragm always at the initial level. The amount of air supplied to the can thus gives a measure of the oxygen absorbed out of the air. No spirometer or blower is used in the apparatus.

Some of the results obtained with the student respiration apparatus are given in Table 8. The alcohol burned varied from 1.22 to 3.51 c.c., the duration of the period from 6.83 to 31.42 minutes, and the oxygen used from 82 to 330 c.c. per minute. The determined values varied between 98 and 103 per cent. of the theoretical values, the average for 33 periods being 100.2 per cent. In the last 10 periods, Wilson soda lime was used. The difference in soda limes apparently had no effect upon the accuracy of the results. Theoretically this ought to be true as the determination of the oxygen with the student apparatus depends upon its replacement by measured volumes of dry gas with like experimental conditions as to volume, temperature, and moisture content at the beginning and end of the period.

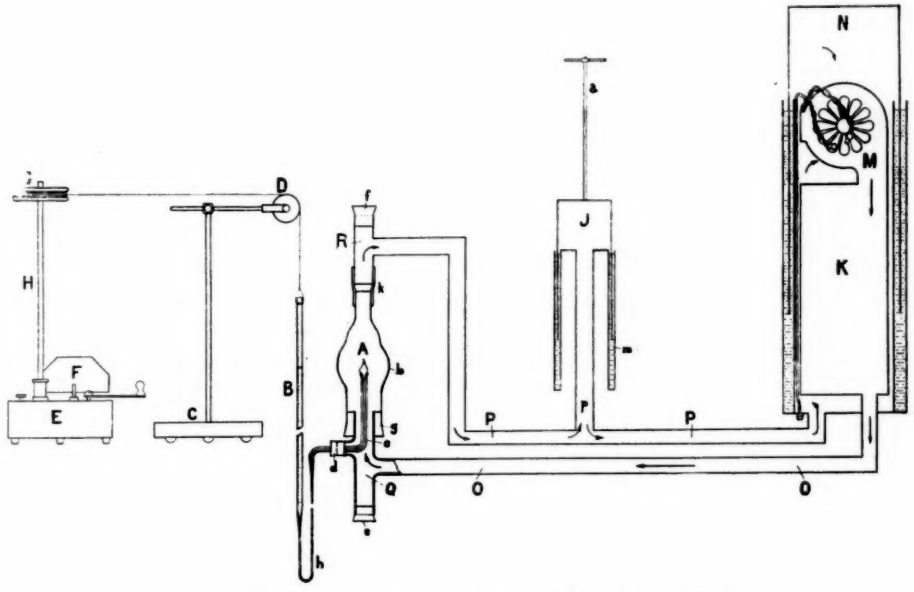

FIG. 1.—Diagram of apparatus used in alcohol check experiments.

A, alcohol lamp in chamber, *b*, with extension, *R*, attached at *k*, and sealed by rubber stopper, *f*. *g*, one-hole rubber stopper, through which is inserted glass tee, *Q*, with side outlet at *d*, and closed by rubber stopper at *e*. *c*, alcohol burner consisting of capillary tube inserted in side outlet, *d*, with wick of asbestos at tip. *B*, graduated burette connected with alcohol lamp by capillary connections, *h*, and actuated by kymograph, *E*. Cord connecting burette and kymograph is attached to pulley *G* on arbor *H*, and runs over pulley, *D*, supported on standard, *C*. Fan, *F*, controls speed of kymograph and regulates flow of alcohol. *m*, water seal for bell, *J*, of hand spirometer; *a*, handle for up and down movement of spirometer bell; *p*, opening for passage of air to and from hand spirometer. *N*, bell of spirometer for Benedict and Collins respiration apparatus; *K*, soda-lime container; *M*, blower (hair-drier) for keeping air in motion; *O*, *O*, pipe for outgoing air; *P*, *P*, for incoming air. Arrows indicate course of air current with Benedict and Collins respiration apparatus.

GENERAL DISCUSSION OF RESULTS

From the preceding series of tests with various types of portable closed-circuit respiration apparatus for the measurement of the oxygen used, it is apparent that no one of the apparatus gives results which are more than 5 per cent. in error, and that, with proper conditions, results can be obtained which, in the majority of the tests, are within 1 per cent. It is also apparent that the conditions which give results with the highest degree of accuracy are those when the apparatus has the same volume at the end and the beginning of the period and the measurement of the oxygen used is either by means of a weighed cylinder of the gas or by an accurate volume measurer such as that secured with the student apparatus; theoretically, also, the best form of apparatus is that in which the air containing carbon dioxide passes through the absorbent before it reaches the contracting volume, *i.e.*, that of the air in the spirometer bell.*

Extensive studies of the moisture conditions inside the bell show, also, that these are more uniform with the Wilson soda lime than with the soda lime heretofore used. The Wilson soda lime develops less heat in use than does the other soda lime, which is of advantage, as with its use there is no uncertainty as to the temperature conditions in the closed-circuit system. The determination of the average temperature conditions and the changes in temperature is one of the most difficult problems in using closed-

*Krogh (Wien. klin. Woch., 1922, xxxv, p. 290) has recently constructed an apparatus for measuring oxygen consumption only, using a closed circuit, a wedge-shaped spirometer, a soda-lime container, and a pair of valves to separate inspired and expired air. M. Krogh and Rasmussen (Wien. klin. Woch., 1922, xxxv, p. 803) have made a comparative study of the results obtained with this apparatus and those obtained with a gas meter for measuring expired air, together with gas analysis. They find that the Krogh apparatus gives systematically 2 per cent. lower results than the combination of gas meter and gas analysis.

circuit apparatus for the measurement of oxygen consumption.* The difference between the heating effects of the two soda limes was determined in studies in which comparisons were made between the temperatures of the air in the spirometer bell, of the air in the pipe leading out from the space surrounding the soda-lime container, and of the air in the room. With the regular soda lime the temperature of the air in the outgoing air pipe was, in some cases, lower at the beginning of a series than the temperature of the air in the spirometer bell, but at the end, the temperature of the air in the outgoing pipe was higher than that in the bell, even though the latter had also risen. With the Wilson soda lime, the temperature of the air in the outgoing pipe was nearly the same, or slightly lower, than that of the air in the bell and the changes in both temperatures were parallel. Subjectively, the difference was also noticed by handling the container. With the regular soda lime there was a marked heating effect, but with the Wilson soda lime the change was hardly appreciable. The difference in the heating effect is due to the difference in the absorption and vaporization of moisture. With the ordinary soda lime there is a heating effect due to the absorption of carbon dioxide and also to the absorption of water by the caustic alkali. With the Wilson soda lime there is a lower percentage of caustic alkali, and any heating effect due to the absorption of carbon dioxide by the lime is offset by the vaporization of water from the soda lime itself. In addition, there is sufficient water in the Wilson soda lime to keep the air saturated to the same degree, whereas with the ordinary soda lime the saturation varies according to the length of use and the amount of water the lime has absorbed.

A word of caution regarding the use of the Wilson product should be given here. As supplied, this soda lime is in lumps of smaller size than the regular soda lime; consequently the container should not be filled to such an extent as to interfere with the ventilation through the apparatus.

SUMMARY

A series of ethyl alcohol check tests has been carried out with the Benedict and Collins portable respiration apparatus, ordinary soda lime being used. The calculations were made on the assumption that the gas in the spirometer bell was dry. The correction for rise in temperature was calculated to be 1.8 mm. for each degree Centigrade and was applied to the whole contraction of the spirometer bell instead of 1.8 c.c. per minute per degree being used as recommended by Benedict. The new correction is not significantly different in final results when the periods are about 12 minutes in duration, but is more exact when the periods are of varying length. An average error of +4 per cent. was found. An extensive experimental study has shown that the error is made up of about 0.5 per cent. due to the presence of carbon dioxide in the air in the spirometer bell, about 2 per cent. due to the moisture content of the air, and the remainder to error in the measurement of the absolute temperature of the air in the whole apparatus. When Wilson soda lime was

Table 7. Results of alcohol check tests with the Roth modification of the portable respiration apparatus.

Date.	Duration.	Alcohol burned.	Rise in temperature.	Oxygen used per minute.	Per cent of theory.
	min.	c.c.	°C.	c.c.	c.c.
1923					
Jan. 17	10.0	2.53	1.4	276	101.0
	10.0	2.58	0.4	279	99.5
	10.0	2.54	1.0	275	100.0
	10.0	2.16	-0.2	239	102.0
	10.0	1.90	2.4	205	99.5
	13.0	1.90	1.0	158	100.0
	11.0	1.43	0.7	143	101.5
	14.0	1.67	0.5	129	99.5
Apr. 16	15.0	2.95	-0.5	210	98.5*
Apr. 18	15.0	3.16	-0.3	237	99.5*
	15.0	2.91	0.8	210	99.5*
	15.0	2.72	1.6	199	101.0*
	15.0	2.75	2.1	199	100.0*
Apr. 20	15.0	2.88	-0.2	206	99.0*
	15.0	2.68	0.1	192	99.0*
	15.0	3.04	1.5	222	100.5*
	15.0	1.87	1.5	136	101.0*
	15.1	1.82	0.8	134	103.0*
	15.0	2.90	1.4	208	99.5*
	15.0	1.85	1.4	133	99.0*
	15.0	1.87	1.2	137	101.5*
	15.0	1.65	1.1	120	100.5*
Average					100.2

* Wilson soda lime was used and a 2 per cent correction made in calculating the results.

*This difficulty was very clearly pointed out by Benedict in the first description of the portable apparatus.[13]

used and the reduction to 0° and 760 mm. was calculated on the assumption that the air was completely saturated, the results were very satisfactory, this group of experiments giving an average of 100.3 per cent.

A similar series of tests with the Roth modification of the apparatus gave good results with either the regular soda lime or the Wilson soda lime, with an average in these tests of 100.2 per cent.

Table 8. Results of alcohol check tests with the student respiration apparatus.

Date.	Duration.	Alcohol burned.	Oxygen used per minute.	Per cent of theory.
	min.	c.c.	c.c.	%
1922				
Dec. 2	8.92	1.74	211	99.5
	10.62	1.74	178	100.0
	13.61	1.66	133	100.5
	12.88	1.49	128	101.5
	14.05	1.67	127	98.5
	15.00	1.58	114	99.5
	14.28	1.22	91	98.0
	20.28	1.53	82	99.5
1923				
Jan. 24	15.20	1.76	128	102.0
	13.02	1.74	150	103.0
	11.33	1.83	171	98.0
	11.23	1.76	172	101.5
	6.83	2.04	330	102.0
	7.87	1.76	245	101.0
Apr. 12	9.92	1.79	196	100.0
	9.58	1.79	203	100.0
	9.92	1.79	195	100.0
	9.67	1.78	200	100.5
	9.75	1.80	199	99.5
	11.25	1.76	172	101.0
	9.92	1.78	195	100.5
	31.42	3.51	123	101.5
	16.17	1.79	119	99.5
	8.25	1.78	232	100.0*
	9.17	1.79	209	99.0*
	9.00	1.77	212	100.0*
	9.83	1.76	194	100.5*
	9.67	1.77	198	100.0*
	9.50	1.78	205	99.0*
	——	1.75	——	101.0*
	——	1.79	——	98.5*
	——	1.75	——	101.0*
	——	1.78	——	99.0*
Average				100.2

* Wilson soda lime was used

A series of alcohol check tests was also carried out with the student respiration apparatus devised by Benedict and Benedict. These tests likewise gave results with a high degree of accuracy with both forms of soda lime. The average for this group of experiments was 100.2 per cent.

In conclusion we make the following recommendations:

1. That with the Benedict and Collins portable respiration apparatus the correction for change in temperature of the bell be made on the whole contraction as a rise of 1.8 mm. per degree Centigrade; and that Wilson soda lime be employed as the absorbent for carbon dioxide, and the assumption be made, in calculating the results, that the air in the spirometer bell is completely saturated.

2. That users of the Roth modification of the Benedict and Collins portable apparatus calculate their results on the basis that the air is dry when the ordinary soda lime is used and employ Roth's correction of minus 2 per cent. when Wilson soda lime is the reagent for absorbing the carbon dioxide; and that the correction for temperature be made on the whole contraction as directed by Roth, viz., 0.5 mm. per degree Centigrade.

3. That whether the ordinary soda lime or the Wilson soda lime is used with the student apparatus of Benedict and Benedict, the calculations be made on the basis that the gas is in a dry condition.

We desire to express our thanks to Professor Francis G. Benedict for suggestions and helpful criticism in the carrying out of these investigations.

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A NOTE ON THE FALLACY OF USING ALCOHOL FOR THE STERILIZATION OF SURGICAL INSTRUMENTS

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AND
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On October 18, 1921, a young woman, age 17, was admitted to the Second Surgical Service of the Boston City Hospital for observation. Her history was negative except for a rather large, fluctuant vascular tumor situated underneath the skin covering the left scapula. This mass had been present for several years, at least. The skin was intact. A pre-operative diagnosis of hemangioma was made, and operation advised. On October 22, under ether anesthesia, the tumor was removed. Because of the nature and size of the tumor rather extensive dissection was necessary, resulting in con-

siderable loss of blood and postoperative shock. The incision was tightly closed with silkworm gut sutures and a small rubber tissue drain was inserted into a stab wound below the incision. Dry sterile dressings and a pressure bandage were applied. Because of the persistent state of shock she was transfused the next day. She died the morning of October 24, approximately forty-eight hours after operation. Autopsy revealed crepitant infection of the subcutaneous tissues and superficial muscles of the back and left side of the abdominal wall, extending down as far as the thigh. Smears from the wound showed small Gram-positive bacilli with occasional spores. A dextrose agar stab culture, which before inoculation had been overlaid with sterile melted vaseline, heated to 100° C. for ten minutes and cooled, showed abundant gas after forty hours incubation at 37.5° C. Smears made from this culture and stained by Gram's method showed Gram-positive spore bearing bacilli with rounded ends. This stab culture was heated at 70° C. for ten minutes, cooled and subcultured to another similar dextrose agar stab and also to a deep tube of plain bouillon previously overlaid with vaseline, heated to 100° C. and cooled. On incubation both showed growth with gas within twenty-four hours, and stained smears showed Gram-positive bacilli identical with those described above. Five cubic centimeters of the plain bouillon culture were injected into the ear vein of a rabbit. The rabbit was killed in ten minutes and placed in the incubator at 37.5° C. for four hours. The result was typically that caused by B. aërogenes capsulatus, or one of the closely related gas producing sporulating anaërobes, namely: general subcutaneous crepitus, enormous abdominal distention, and gas bubbles throughout the muscles, blood vessels, liver and other viscera with smears showing only large Gram-positive encapsulated bacilli.

On October 23, 1921, a man, age 44, was admitted to the Third Surgical Service with a history of severe right lower quadrant abdominal pain of two days' duration. A diagnosis of acute appendicitis was made, and he was operated on a short time after admission. Under ether anesthesia a large gangrenous appendix was removed with difficulty; the peritoneal cavity contained seropurulent fluid. Two cigarette drains were inserted, the abdominal wall was sutured in layers, and an alcohol dressing applied. Two days later a tender light brown area appeared just above the crest of the right ileum; this increased rapidly in size. The edges of the skin incision were bluish black, but no crepitus could be made out. That afternoon the tender area was incised, and the operative wound was laid open to the fascia; both were packed with H_2O_2 gauze. The next day he was delirious, the discolored area had increased greatly in size, and he died about noon, approximately seventy-two hours after operation. Smears from the incision wound made October 25 showed large Gram-positive spore-bearing bacilli with rounded ends. A plain agar stab culture, which had been overlaid with sterile melted vaseline, heated to 100° C., and cooled, showed gas after twenty-four hours incubation at 37.5° C., and on smear large Gram-positive bacilli with rounded ends. This stab culture was heated to 65° C. for forty-five minutes and transplanted to another similar dextrose agar stab; the latter showed gas in twenty-four hours, and on smear large Gram-positive bacilli with rounded ends. The specimen was reported "B. aërogenes capsulatus."

The condition of two other postoperative wound infections was such as to warrant bacteriological examination for the presence or absence of B. aërogenes capsulatus. These were sent to the laboratory October 26 and 27, and were both reported negative.

In view of two deaths within forty-eight hours from gas bacillus infection, one of which was a "clean" case, and of two other suspicious cases, the operating rooms were closed except for emergency operations. An attempt was made to locate the source of infection. Anaërobic and aërobic cultures of gauze, catgut, rubber gloves, basins, bandages, and rubber drains were sterile. One Bard-Parker knife blade was taken direct from the instrument cabinet and placed in a deep tube of plain bouillon, which previously had been overlaid with sterile melted paraffin, heated to 100° C., and cooled. After twenty-four hours incubation at 37.5° C., there was abundant growth with gas formation. Stained smears showed large Gram-positive bacilli with rounded ends, along with other organisms. The tube was heated to 70° C. for ten minutes and a transplant made to a plain agar stab, which had been covered with sterile melted vaseline, heated to 100° C. and cooled. The subculture showed abundant gas after incubation for twenty-four hours, and on smear Gram-positive sporulating bacilli with rounded ends. A transplant was made to an anaërobic plain bouillon tube, and after twenty-four hours incubation several cubic centimeters were injected into the ear vein of a rabbit. After killing the animal and incubating the carcass, the result was typically that produced by B. aërogenes capsulatus or one of the organisms belonging to that group.

A canvass of the surgical records revealed the following: On October 14 a man was admitted to the First Surgical Service with the diagnosis "Gunshot wounds (both legs) and compound fracture (fibula) of left leg." Shortly after admission and under ether anesthesia the bullet was removed from the right leg and the macerated tissue removed from both wounds as far as possible. The wounds were flushed with salt solution, H_2O_2, and finally alcohol. A strip soaked with iodin was drawn through the wound in the left leg and alcohol dressings were applied. On October 18th the left leg became con-

siderably swollen, reddened, and painful. A culture taken for bacteriological examination was reported "B. aërogenes capsulatus." On October 20th the patient was taken to the operating room, and both the wound of entrance and exit were opened up wide. They were flushed out with salt solution and alcohol, a Dakin's tube was inserted, and a posterior wire splint applied. Swabs taken at the time were reported positive for B. aërogenes capsulatus. Recovery was uneventful, and a swab taken November 1st was reported negative for B. aërogenes capsulatus.

As a matter of operating-room routine all scalpels, scissors and Bard-Parker knife blades had always been washed thoroughly in hot soap and water, rinsed in scalding water, and then stored away in the instrument cases. Before using again they were placed in sterilizing solution (70% alcohol) for five to twenty minutes.

In order to determine the effect of the operating room sterilizing solution (70% alcohol) on a gas-producing sporulating anaërobe, the following experiment was performed. Six six-inch pieces of fairly heavy sterile iron wire (about 18 gauge) were immersed in an anaërobic plain bouillon culture of the gas-producing sporulating anaërobe obtained from the Bard-Parker knife blade. The wires were withdrawn, placed in a large sterile test tube, and incubated at 37.5° C. until dry. Five were then placed in individual sterile test tubes containing sterilizing solution, and allowed to remain 5, 10, 15, 30, and 60 minutes. The five wires and the one untreated wire, serving as a control, were cultured anaërobically in plain bouillon. All showed abundant growth and gas after twenty-four hours incubation at 37.5° C.

It seemed logical to conclude that in all probability at least one, and possibly two, of the fatal "gas" cases were due to infection at the time of operation by Bard-Parker blades or scissors used two or three days previous on a known "gas" case, and improperly sterilized prior to re-use.

In view of these findings it seemed advisable to change the method of operating-room sterilization of scalpels, scissors, and Bard-Parker blades. It was decided that they should be sterilized along with the other instruments, that is, by immersion in boiling water for twenty minutes. In addition it was decided to discard all Bard-Parker blades after use. Operating was resumed and since that time no cases of postoperative infection have occurred in which suspicion could be directed to the improper sterilization of instruments as the source of contamination.

From the simple experiment reported herein it can be definitely stated that immersion in alcohol (70 per cent.) for one hour will fail to sterilize instruments grossly infected with bacilli belonging to the B. aërogenes capsulatus group.

MILK AND THE PUBLIC HEALTH

BY JOSEPH GARLAND, M.D., BOSTON

III. Past, Present and Future

HISTORICAL

The production and distribution of milk have been subjects of discussion for certainly nearly thirty years in this Commonwealth; perhaps ever since the vexing problems of supply and demand, of transportation and consumption, made large-scale operations and middlemen's profits a factor in our economic problems, and substituted for the person-to-person transaction the complexity of modern business methods. This discussion was at first purely economic and engaged in by producers, contractors and transportation agents.

When, in 1906, the first systematic dairy inspection was initiated by the State Board of Health, considerable criticism was aroused. The health question was then more pointedly injected into the discussion, and the publicity given to milk as a source of disease was branded by many dairymen as an injustice which tended to injure their business. This has been to some extent the attitude which many of them have maintained since then, though I believe to a lesser degree. Efforts to bring together apparently opposing interests such as the State Department of Public Health, the State Board of Agriculture, the Grange, and the dairymen have not been very successful. Local milk inspectors also have opposed legislation tending to centralize authority of milk inspection.

All in all the discussions have been participated in by the Boston Chamber of Commerce, the Grange, the State Board of Agriculture, the Department of Animal Industry, the Milk Consumers' League, labor organizations, Milk and Baby Hygiene Associations, contractors, medical societies, and other interests.

Already, previous to 1915, three investigations into the milk situation had been conducted, and the remedies proposed by all were based on increased legislation; in fact, in the years from 1910 to 1914 inclusive 34 separate laws were proposed designed to improve the production and distribution of milk. In the main, however, bills introduced for the protection of the consumer and which might tend to increase the cost of milk production were difficult of passage. For several years prior to 1911 Dr. Charles Harrington, former secretary of the State Board of Health, introduced bills in the Legislature which would enable the State Board of Health to enforce its suggestions for cleanliness in the dairies supplying Massachusetts. These bills could not be passed.

The Massachusetts Milk Consumers' Association was formed in 1910, its purpose being "An association formed to unite consumers in obtain-

ing efficient inspection and a pure milk supply." This Association in 1911 proposed, and supported the Ellis bill placing the administration of milk regulation in the hands of the State Board of Health. The bill was passed but was vetoed by Governor Foss, who stated that "What we need is to penalize the farmer a little less and encourage him a good deal more." There is probably more than a kernel of truth in this statement, despite the fact that it accompanied the vetoing of a commendable bill.

PRESENT LEGISLATION

Our present Massachusetts dairy laws are voluminous after the fashion of legal documents. In a nutshell it may be stated, however, that the Massachusetts legal standard for milk consists of a milk which upon analysis is shown to contain not less than 12 per cent. of milk solids and not less than 3.35 per cent. of milk fat. Grade A milk must be produced within the Commonwealth from healthy cows under cleanly and sanitary conditions, and so cooled and cared for that in its raw state the bacteria count shall not average more than 100,000 per cubic centimeter, upon examination of five samples taken one each day, and each from a different lot of milk, on five consecutive days. This shall be labeled "Grade A. Massachusetts Milk," etc. The per cent. of milk fat may be stated but shall never be less than the standard fixed for milk.

The fact is worth noting that Grade A is the only milk on which direct legislation imposes a maximum bacteria count, and that no milk is required to be pasteurized, although a legal definition of pasteurization is provided. It is also interesting to note that a penalty is attached to heating milk to more than 167° F. without labeling it as such, a procedure which, above all others, would safeguard that milk!

It is of interest to compare these regulations with those in force in New York. The system of milk grading in New York City had been in operation for three years, when the report of the Milk Board was published in 1915, and had been meeting with success. Four grades are provided for, varying in their price. Grade A raw milk, the only milk to be sold raw, must come from tuberculin tested cows, must be produced under conditions similar to certified milk, and the bacteria count is limited to 60,000 per cubic centimeter. Grade A pasteurized milk need not come from tuberculin tested cows, but the allowable bacteria count is limited to 200,000 before and 30,000 per cubic centimeter after pasteurizing. Grade B, pasteurized only, may have a bacteria count of 1,500,000 per cubic centimeter before pasteurizing in the city, 300,000 before pasteurizing in the country, and 100,000 after pasteurizing. Grade C, pasteurized, may have a maximum bacteria count of 300,000 and must be labeled, "For Cooking Purposes Only." Milk may be pasteurized only once.[4] These regulations, incomplete as they are, are, nevertheless, far superior to our present ones in Massachusetts.

The great argument used by the opponents of pasteurization is that it encourages the production of dirty milk and careless handling. For this reason, in mentioning pasteurization in these pages it has generally been qualified by the words "proper" or "efficient." "Proper" pasteurization entails the pasteurization, according to legal standards, of a fresh milk, cleanly produced and cooled after milking and after pasteurization. If these processes are efficiently overseen pasteurization is a valuable protection and not the whitewash for a sepulchre.

TABLE 1.—Growth of bacteria when milk is held at 50° F. and at 68° F.

Temperature of milk (°F.).	Number of bacteria per cubic centimeter.				
	At beginning.	At end of 6 hours.	At end of 12 hours.	At end of 24 hours.	At end of 40 hours.
50	10	12	15	41	62
68	10	17	242	61,280	3,574,990

Most of our legislation, as has been indicated, deals with the butter fat and solids content and the gross cleanliness of milk; standards which were unquestionably considered of major importance a few years ago. That they are of importance no one would care to deny, for no one wishes to buy an adulterated food or an impoverished one. The health problem, however, depends on the bacterial content and, of course, the qualitative rather than the quantitative factor is the important one. There would be no great difficulty experienced in choosing between a milk containing 1,000,000 lactic acid bacilli per cubic centimeter and one containing 1000 typhoid bacilli. The suppression of bacterial growth by the proper cooling of milk is well exemplified by the preceding table.[14]

Massachusetts, although tardy in her acceptance of bovine tuberculosis eradication work, has at last embarked on it in an apparently businesslike fashion. This late arrival of the Common-

wealth is probably in part due to her earlier experience. About 1895, soon after bovine tuberculosis became an established fact, a compulsory tuberculin test was inaugurated. Tuberculous infection was at that time so prevalent that frequently from 30 to 50 per cent. of the cattle tested were found to be reactors and were destroyed. Our dairy farmers found themselves facing ruin, the tuberculin test was discredited, and the Act was repealed.

By the Acts of 1922, now in effect, owners may have their cattle tuberculin tested by the state inspector and may receive as indemnity for their condemned cattle one-third of the difference between the amount received by the owner for the carcass of the animal and the value of the animal as determined by appraisal, provided that in no case shall payment by the Commonwealth exceed $25.00 for any grade animal or $50.00 for any pure-bred animal. The Federal Government also pays one-third of this difference, and the balance of the loss is borne by the owner. The progress of this eradication work has already been stated.

CERTIFIED MILK

The methods and standards for the production and distribution of certified milk were adopted by the American Association of Medical Milk Commissions at the sixth annual meeting of the Association at Louisville, Kentucky, in 1912.

Certified milk is considered as the product of dairies operated in accordance with accepted rules and regulations formulated by authorized Medical Milk Commissions to ensure its purity and adaptability for infants and invalids.

The need for such a milk, according to the standards then formulated, was experienced primarily by those engaged in the conservation of the life and health of infants. As a result, there was introduced in 1892 a plan whereby certified milk would be produced by a dairyman under the control of a Medical Milk Commission designated by a representative medical society.

The first rules designed were contained in an agreement entered into by a Medical Milk Commission and the dairymen concerned. These first rules represented the essential requirements for the production of certified milk. Subsequently other commissions were organized, which, in 1906, were federated into a National Association, whose fundamental object was "to bring about the uniformity of standards and their perfection." From time to time new standards have been adopted relating to the veterinary inspection of herds and farms, the medical inspection of milk-handling employees, and the bacteriological and chemical examinations as to quality and purity. The rules are extensive and detailed; among the most important provisions are those requiring that all certified milk shall reach the consumer within 36 hours after milking, and that 10,000 per cubic centimeter shall be the maximum bacteria count of the milk when delivered. Provision for Medical Milk Commissions is made in the Massachusetts Dairy Laws.

In Massachusetts there are now nine farms producing certified milk from 699 cows; 4436 quarts are distributed daily at prices ranging from 24 to 35 cents per quart, the average price being 26.7 cents. The cost of producing a quart is close to 20 cents. In some instances it is claimed that certified milk is produced at a loss which has to be borne by the sale of uncertified milk by the same dairy.

The Medical Milk Commissions have served a useful purpose in demonstrating the conditions which should be approximated in the production of all milk, and in awakening an interest in the methods of production of pure milk. Doubtless, the cost of production, excessive as compared with other grades of milk, is in part due to refinements which are not strictly necessary.

The chief practical defects in the production of certified milk as a public health measure are its limited availability and its reliance on the human factor—an efficient Milk Commission, an honest dairyman and intelligent employees—to ensure its purity, in place of sterilization of the milk. To sum it up fairly, the production of certified milk is a valuable and instructive demonstration in showing how well milk may be produced, but it is not a factor of importance in itself in safeguarding the public health.

PRODUCTION AND CONSUMPTION

The comparison of the food value of milk with that of other commodities is an old scheme of milk dealers to demonstrate its superiority. Nevertheless, there is clearly an important lesson to be learned from such comparisons, for milk is certainly an unduly inexpensive food when viewed in this light. According to the Milk Report of 1915 four quarts of milk is the nutritional equivalent of four eggs, three-quarters pound of lean beef, two pounds of potatoes, one pound of cabbage, one-half pound of bread, and one-twenty-fourth pound of butter, easily a day's ration. The comparison of the price of four quarts of milk and the total of the other commodities speaks for itself—and yet on some farms cows are kept for the value of their manure as a fertilizer, the milk being purely a by-product!

The quantity of milk consumed, however, is no mean factor, though it should reasonably be greater. To illustrate this the daily milk consumption of seven representative cities has been secured, including the proportions of the total pasteurized and certified.

Boston.—387,320 quarts sold daily—93.42 per cent. pasteurized; 3581 quarts are certified.

Brockton.—26,600 quarts sold daily—40 per cent. pasteurized; 75 quarts certified.

Fall River.—48,000 quarts sold daily—47 per cent. pasteurized; none certified.

Holyoke.—29,000 quarts sold daily—85-90 per cent. pasteurized; none certified.

Lawrence.—48,900 quarts sold daily—75 per cent. pasteurized; 50 quarts certified.

New Bedford.—60,000 quarts sold daily—55 per cent. pasteurized; none certified.

Salem.—23,000 quarts sold daily—60 per cent. pasteurized; certified not stated.

A large proportion of Boston's milk supply is produced in New Hampshire, Vermont, Maine, New York and Canada. Nevertheless, Massachusetts dairying is coming back, according to the Massachusetts Department of Agriculture, which justly emphasizes the advantage of fresh milk produced near by over milk which is gathered three times a week in distant localities and subjected to a rail journey of many hours before distribution. As an illustration of this hopeful trend of affairs they point out that there has been a 50 per cent. increase in pure bred dairy animals in Massachusetts in the last five years, and that now pure bred Massachusetts cattle are being sent to many parts of the country.

GENERAL CONSIDERATIONS

It is not possible to say and it would be idle to conjecture as to whether or not King Tutankhamen's supposed tuberculosis had its origin in the milk of the cows that grazed along the Nile five thousand years ago. We do know that the disease is as old as history itself, for Hippocrates, if he did not recognize that human tuberculosis might originate with cattle, at least knew and recognized the disease. Bone and joint tuberculosis and scrofula—the king's evil—now more generally known as tuberculous cervical adenitis, have been among the plagues of humanity for many generations. There is no reason to doubt that then, as now, these diseases had their beginnings in many instances in infected cow's milk. It is for this very reason that the popular mind has not long ago demanded an inquiry into the causes of these afflictions, for that which has been always with us arouses little comment. The village hunchback, the scrofulous person, the neighbor's boy with hip disease, the child that died from meningitis aroused our sympathy but not our curiosity; they were afflicted by God, but we did not know that God might have worked through the agency of our own cow, and we did not know that pasteurizing or boiling her milk might have prevented these disasters.

The universal sterilization of milk—the only animal food that is habitually eaten raw—will be the first step in this prevention. Universal eradication of tuberculosis in cattle will be the second step. "The cow reacted but at post mortem did not show disease," has been the frequent complaint of cattle raisers. This, of course, is true, just as it is true that a large proportion of adult human beings are reactors. In both cases it means the same thing—a tuberculous infection, but in the one case we can and must slaughter the reactors in order to build up a tuberculosis-free cattle population that will help us to diminish the tuberculosis incidence in the other case, for the process of human tuberculosis control must be a gradual and slow one, including treatment of the infected, as well as prevention of infection. After all is said and done, human and bovine tuberculosis may well be the same disease, and the two types of bacilli may be the descendants of a common parentage, only altered by environment, as are probably the viruses of smallpox and cowpox.

Legislation, at first directed towards an economic situation, later designed to standardize the chemical purity of our food supplies, is at length slowly revolving towards the far more important and fundamental problem of the prevention of bacterial diseases as borne by food. Pasteur was born one hundred years ago, but our realization of the part that bacteria play in our afflictions and the degree to which the offending organisms may exist in our common foods is in its infancy.

As this knowledge has grown and been appreciated by small groups of individuals their tendency has been to introduce and legalize standards designed for the protection of their fellows. The flaw in this method has been that their fellows were ignorant of the dangers from which they were being protected, and failed to appreciate and were often intolerant of the efforts that were being made in their behalf. It is a popular belief in this country that the people make the laws; it is certainly a fact that those laws which are most effective in their workings are laws which have found their places on the statute books as the result of a general popular demand. Slavery does not exist because the people would not tolerate it. Prohibition exists largely in name only, because the people are tolerant of its failures.

Obviously, then, since laws of which we are ignorant and laws of which we fail to see the value are often lacking in desired results, it must be our duty to help create a popular demand for what we know is good, and that demand will be supplied, whether by proper legislation, supported by the people; or by the force of public opinion alone.

With this popular demand must come education to an appreciation of the value of what we are demanding, for satisfaction must be maintained along the line from producer to consumer. Milk, despite its slight increase in cost over former years, is still by far the cheapest food we use; if we are not getting safe milk we are not paying for safe milk. If we demand

safe milk of the producer we must show our willingness to pay for it, and he will be willing to supply it. Our failures in the past have been because, while we demanded pure milk as we knew it, we were insisting that the extra burden of producing it be borne at the source. The increased payment must be an accompaniment of the increased quality, for the farmer's business must be protected in order to insure him his deserved independence.

A popular demand for the eradication of bovine tuberculosis, then, must be part of our program. Hand in hand with this must go a popular demand for milk cleanly produced and effectively sterilized, and a willingness to pay a reasonable price for it.

In closing I will quote a few lines from the Report of the Special Milk Board:

"To insure its safety as a human food two procedures of control are necessary:

"First.—By inspection of cattle, methods of production and transportation, secure a supply that is pure, clean and fresh.

"Second.—By the application of heat, destroy all disease germs that may be in it, whether they come from animal or human sources.

"To obtain the most perfect results market milk, intended for human consumption as milk, should be pasteurized under the control of central authority and in the final container."

REFERENCES

4 Report of the Special Milk Board of the Mass. State Dept. of Public Health, Dec., 1915.
14 Mass. State Dept. of Agriculture: Bulletin No. 38.

The author acknowledges with thanks the valuable assistance of the following, who have furnished much of the material used in these articles:

Mr. O. M. Camburn, Director, Division of Dairying and Animal Husbandry, Massachusetts State Department of Agriculture; Dr. Bernard C. Carey, Director, Division of Communicable Diseases, Massachusetts State Department of Public Health; Dr. E. A. Crossman, Federal Inspector in charge of Bovine Tuberculosis Eradication, Customs House, Boston; Dr. Lester H. Howard, Director, Division of Animal Industry, Massachusetts State Department of Agriculture; Mr. Hermann C. Lythgoe, Director, Division of Food and Drugs, Massachusetts State Department of Public Health; and the Health Departments of Boston, Brockton, Fall River, Holyoke, Lawrence, New Bedford and Salem, which responded to a questionnaire.

Book Reviews

A Manual on Ship Sanitation and First-Aid for Merchant Seamen. Prepared under the direction of the Rev. Archibald R. Mansfield, D.D., Superintendent, Seamen's Church Institute of New York, in coöperation with the United States Public Health Service, Washington, D. C., by Robert W. Hart, Passed Assistant Surgeon. Second edition. With illustrations. Published by the Seamen's Church Institute of New York. Price $1.00.

This little 5 x 7½ inches book of 172 pages attempts to cover General Sanitation and Hygiene, especially as related to conditions on shipboard, Anatomy and Physiology, Ship's Sick Bay and Medicine Chest, Medical First Aid, Surgical First Aid, Regulations U. S. Public Health Service.

The only paying merchant marine this country ever had was built up under a simple system of shipboard therapeutics which regarded a sailor as either fit to work or sick enough to take calomel and jalap. Sailors and their advisers were, however, not content to let this system alone, and such additional therapeutic agents as Brown's Bitters and Squibbs Cholera mixture came to be favored for symptoms resulting from getting overloaded ashore with alcohol, unaccustomed food or polluted water, or in painful internal derangements which sometimes included appendicitis or dysentery, and quinine became the approved medicament for every ailment in which fever appeared as a conspicuous symptom.

As no noteworthy improvement in health conditions on ships was brought about by thus complicating shipboard medicine, further attempts have been made from time to time, and this last attempt still refers to conditions as unsatisfactory.

Probably conditions will always be unsatisfactory, but they might be somewhat better if those who have tried to improve them had a better appreciation of actual shipboard needs. Attempts have been made in this little book to crowd too much into too small a space. Dogmatic instructions presented without a rational basis are more likely to be confusing than helpful in emergencies.

Ship masters used to attribute to one another the story of the ship captain who had supplied himself with a ship's medicine chest in which the medicines were numbered and the chest accompanied by a book in which certain groups of symptoms were described and the proper medicine for each group indicated by its number. On his return from a voyage the captain complained to the druggist who sold the chest that the medicines were "no good." He had had a sick man presenting exactly the symptoms calling for the administration of medicine number eleven, but the man had died. Efforts of the druggist to uphold the reputation of his medicine chest brought out the admission by the captain that he happened to be all out of medicine number eleven, and had therefore mixed number seven and number four together.

There is undoubtedly a place for a book on personal hygiene written to meet the needs of the seafaring man of the type that the missionary or marine hospital doctor knows best. There is also a need of some comprehensive book which will enable a successful ship master to meet medical or surgical emergencies rationally. It can be safely assumed that a man who possesses the intelligence to become a successful ship master can understand anything that is really under-

stood in medicine or surgery, provided it be presented in an understandable way and provided the ship master find himself confronted with the necessity of understanding it. This little book in attempting to fill both these needs fails to fill either.

On ships on which health instruction is needed most it may be assumed that the master himself will have to see that everything is done properly, from keeping the ship decently clean to attention to a compound fracture of a leg. It is appropriate surgical treatment for injuries which is most important on shipboard. In the long run probably the less that is tried on shipboard in the way of treatment for sick men the better it will be for them. A book intended to promote the health of seafaring men ought to emphasize the advice to do nothing either in sickness or accident until sure that the right thing is being done.

The captains of our old Arctic whaling fleet of forty years ago often did excellent emergency surgery in injuries resulting in fractures and dislocations. There were always to be found in the cabins of such captains the latest works on anatomy and surgery. It is true that very discreditable examples of emergency surgery on shipboard are admitted to marine hospitals, but ship masters or mates are not responsible for all. Some are the handiwork of men on ships who hold degrees of Doctor of Medicine.

The little book under review purports to instruct in emergency surgery—its practical value for this purpose is small. What is really needed is—to take the subject of fractures and dislocations, for example—brief, concise instructions explanatory to comprehensive diagrams and reproductions of photographs showing surgical landmarks, methods of measurement, characteristic deformities resulting from fractures and dislocations, muscular attachments, and causes of such deformities and rational methods of reduction. In other words, something which will enable a ship master to act rationally in surgical emergencies and comparable to the plans, diagrams, specifications and instructions which it is deemed necessary to supply to the officers of a modern ship to enable them to understand and care for the ship itself and its machinery. Such a book cannot, of course, be sold for a dollar.

But, even within the compass of this little book, it would seem that opportunity might have been found to say something about the practical value of gasoline in helping to clean certain kinds of wounds, or to add a caution as to the danger of incomplete chloroform anesthesia, or to mention ether as an anesthetic, or to say something more about the care of compound fractures, or to call attention to the great importance of the immobilization and rest of an injured part in enabling the tissues to prevent the development of an infective process and to check the extension of such a process even after it has started.

The Operative Treatment of Glaucoma. By Lt.-Col. H. Herbert. New York: William Wood & Co. Pp. 152. Price $3.00.

The last few years have been marked by the unusual interest of the profession in the problems of glaucoma, as evidenced by the publications on the subject: The second and enlarged edition of Elliot's very sound treatise, the volumes of Morax and of Lagrange, and now this monograph by Colonel Herbert.

The practical introduction of the fistulizing operation by Lagrange was a great step in advance, but all operations of this type have their drawbacks, and many have been the modifications proposed and tried. For twenty years Herbert has been working and writing on this problem, and in this work of nearly 150 pages he gives us his conclusions and the reasons therefor.

For the milder cases of glaucoma he recommends several procedures: A subconjunctival series of scleral incisions resembling a garden rake in form, the subconjunctival excision of a wedge of sclera. For the more severe type of the disease Herbert is the enthusiastic advocate of some type of the iris inclusion operation by which a greater or less amount of iris tissue is included in the wound (under a conjunctival covering) and thereby tends to permit filtration of the intraocular fluid. Vericular conjunctival patches should, in his opinion, become a thing of the past, on account of the danger of later infection. For this reason he is against the Elliot trephining operation.

The last thirty pages are devoted to a description of the operative technique of his personally devised methods.

A Simple Treatment for Tuberculosis. By Owen F. Paget, M.D. New York: William Wood & Company. Price, $1.75.

In this little volume of approximately 75 pages Dr. Paget advocates a new and interesting method of using tuberculin in the treatment of all forms of tuberculosis. This treatment consists of the insufflation of a preparation containing dead tubercle bacilli into the nose. He believes that the nasal mucous membrane has a special power of developing tuberculous antibodies and on this belief he bases his method of treatment.

He reports numerous patients who have apparently shown remarkable improvement as a result of this treatment.

It is manifestly impossible to come to any definite conclusions on the efficacy of this method of using tuberculin as advocated by Dr. Paget on his word and experience alone. To those

of us who have used tuberculin in suitable cases and who have watched its effects, usually disastrous, on pulmonary lesions a somewhat skeptical attitude is the natural one to take.

I find myself utterly unable to believe that tuberculin, whether in the form of a bacillary emulsion or a bouillon filtrate or any other preparation taken into the body by the means of the nasal mucous membrane, can differ in any way in its effect from the same preparation introduced subcutaneously or otherwise.

Until, therefore, further proof is presented as to the reliability and harmlessness—and particularly the harmlessness—of this means of using tuberculin,—an agent in the hands of the untrained most potent for harm,—the medical profession as a whole might well leave this procedure alone for the present at least.

Maladies del' Appareil respiratoire. Tome II du Precis de Pathologie Médicale. Par MM. F. Bezancon, Professeur à la Faculté de médecine de Paris, et S. I. de Jong, médecin des hopitaux de Paris. 1 vol. de 566 pages, avec 83 figures et 2 planches en couleur. Collection de Precis Medicaux (Masson et Cie., Editeurs). Broché .... 20 fr. Cartonne toile .... 25 fr.

This is the third volume of the projected six which will comprise the Precis de Pathologie Médicale. The other two volumes, one on the diseases of the blood and hematopoietic organs and the diseases of the kidney, the other on diseases of the digestive tract and diseases of nutrition, have already been reviewed. This volume deals with diseases of the respiratory apparatus and is by Fernand Bezancon, and S. I. Jong.

The volume, in spite of its small size, is very complete. It begins with chapters devoted to the anatomy and general pathology of the respiratory tract. Then follow the diseases of the nose, larynx, trachea, bronchi and lungs. There is a small number of illustrations admirably chosen to illustrate the pathology and anatomy. In spite of the French designation, "Pathologie Médicale," the book is essentially a treatise on clinical medicine. The reviewer wishes to comment particularly on the treatment of mechanical lesions of the lung, such as bronchiectasis and emphysema.

For medical students and physicians who have use for a most admirable compendium and who wish to practise reading French, the volume cannot be recommended too highly.

Principles of Bacteriology, second edition. Eisenberg. Published by C. V. Mosby Company, St. Louis.

This little volume is, according to the preface, based on lectures by the author, given to nurses. It is appropriately simple in presentation and shows evidence of great care in the selection of topics. It is fairly complete and shows great wisdom in the elimination of details. It seems to be a text-book admirably adapted to present desirable bacteriological knowledge for nurses and also to gratify the curiosity of nurses in regard to many procedures in hospital practice.

The reviewer commends the inclusion of chapters on immunity, the making of culture media and description of immune phenomena as applied to diagnosis. It is possible to take exception to a few statements and to point out a few mistakes. The statement that the bacterium described by Plotz "has been definitely proved to be the true cause of typhus fever" must be regarded as a bit of carelessness in a text-book revised in 1923.

Les Colites. By Paul Carnot, Professor of the Faculty of Medicine of Paris; Paul Harvier, Physician of the hospitals of Paris; R. Friedel, Consultation Assistant at the Hospital Beaujon; L. Ardennois, Professor of the Faculty of Medicine of Paris. Paris: J. B. Baillière et Fils, 1923.

This book contains concise descriptions of the various forms of digestive disorders in which lesions or functional derangements of the colon play a part. Their etiology and treatment is logically discussed. The work should prove useful to students as a text-book and to practitioners as a book of reference.

Die Experimentelle Bakteriologie und die Infektionskrankheiten mit besonderer Berucksichtigung der Immunitatslehre. A text-book for students, physicians and medical functionaries. By Dr. W. Kolle, Director of the Institute for Experimental Therapy, Honorary Professor in the University at Frankfurt a. Main; and Dr. H. Hetsch, Professor Institute for Experimental Therapy, Frankfurt a. Main. Sixth edition. Berlin, Vienna: Urban & Schwarzenberg, 1922.

The sixth edition of this well-known standard work on the infectious diseases has recently come to hand. It shows evidence of painstaking revision and is likely to fill as useful a place as its predecessors.

Lehrbuch der Grenzgebiete der Medizin und Zahnheilkunde. For students, dentists and physicians. By Dr. Julius Misch, dentist in Berlin. Third edition. Berlin: F. C. W. Vogel, 1923.

The first edition of this book was reviewed about a year ago in the Journal and was commended to the attention of our readers. The appearance of a third edition so soon after the first indicates that the book has already found a useful place. The current edition has been revised and somewhat enlarged.

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THE CASE HISTORIES OF THE MASSACHUSETTS GENERAL HOSPITAL

IN the issue of July 12, 1923, reference was made to the Cabot Case Histories. These have been published by the Massachusetts General Hospital, and have had a world-wide circulation. The BOSTON MEDICAL AND SURGICAL JOURNAL has arranged with the Massachusetts General Hospital to publish these Case Histories as a special feature or department of the JOURNAL, thereby giving to our subscribers the regular JOURNAL and, in addition, reports of clinical exercises that have been recognized as valuable contributions to medical literature.

The Case Histories will continue to present the problems encountered by practitioners in dealing with unusual forms of disease and, in addition to the record of symptoms, clinical and laboratory findings, will be followed by an analysis of all the features found in each case.

These Case Histories are reports of exercises conducted under the supervision of Dr. Richard Cabot and his associates. We expect that the profession will appreciate the value of this type of literature, for the ability and standing of one of the foremost teachers in medicine and the resources of the Massachusetts General Hospital will give to these reports an assurance of correct and modern methods of investigation and treatment.

It should be recognized that the publication of this JOURNAL by the Massachusetts Medical Society is for the purpose of maintaining medical literature representative of New England men and institutions.

In maintaining this JOURNAL the Massachusetts Medical Society is not engaged in a profitable mercantile enterprise, but is spending thousands of dollars more than is received from subscribers and advertisers. The general custom among the state medical societies is to publish a monthly journal. The Massachusetts Medical Society believes in the policy of a weekly journal and hopes are entertained of a coalition of all the New England medical societies for the purpose of developing a journal which will represent this group of States.

Although the publication of the Case Histories will add much to the quality and quantity of reading matter, the subscription price has not been increased.

It is expected that the profession at large will find the JOURNAL worthy of its support.

THE MASSACHUSETTS MEDICO-LEGAL ASSOCIATION

THE fall meeting of this Association was held on October third directly after the Council Meeting. The President, Dr. West, of Newton, in the chair. Previous to the addresses matters of routine business were discussed.

The Secretary brought to the attention of the Society the large number of examiners throughout the State who are not members of the Association and the President asked each member to interview all who were eligible and endeavor to secure a larger membership. Since the by-laws of the Association restrict membership to Fellows of the Massachusetts Medical Society, the President suggested that steps should be taken to amend the by-laws, for it is well known that there are many persons outside the ranks of the Massachusetts Medical Society who are recognized as well qualified and should be asked to join the Association.

The first address was delivered by Inspector G. Gustafson, of the Boston Police Department, who is an expert in criminal investigation. He spoke of the methods used in the identification of human bodies, outlining the earlier use of photographs and the Bertillon system, and explained the inherent defects found in the employment of these exhibits.

He then went on to show that the development of the finger-print method constituted the best known and available system yet devised, for although photographs and bony measurements taken at different periods of life present-

ed variations that are deceptive, the skin ridges on the fingers and hands are permanent from infancy to old age and since no two exhibits have ever been found to be alike the records of finger prints make identification sure and easy. While he acknowledged the value of having records of prints of all persons he conceded the impossibility of bringing about this custom.

Means of identification have great value even outside of criminal investigation, for it has happened many times that important business considerations are concerned in the identification of a human body.

He showed photographs of criminals and finger prints to illustrate his arguments, and his audience showed great interest in the subject under discussion.

The cordial reception accorded to Dr. George B. Magrath, senior medical examiner of Suffolk County, when he rose to speak, demonstrated the esteem in which he is held.

He spoke of the difficulties encountered by the medical examiner outside of Suffolk County because of the control exercised by the district attorneys and explained the dangers of trying to amend the present law which, although defective and unfair to the medical examiners in some ways, had worked fairly well. He felt that a different system of paying for work done by medical examiners would be advantageous, for under the present plan complete investigations may not be approved because of the expense involved.

Although the Society has made especial effort to secure the interest of the medical profession in the work of the medical examiners and the meetings of the Association, very few physicians outside the membership attend the meetings. The addresses and discussions are always informative and interesting to physicians.

NEW ENGLAND SOCIETY OF PSYCHIATRY

On Wednesday, October third, the regular semi-annual meeting of the New England Society of Psychiatry was held in Concord, New Hampshire, the Society being the guest of Dr. C. H. Dolloff, Superintendent of the Concord State Hospital. Before luncheon was served the members were conducted through the wards of the hospital.

At the business session a very interesting program was presented. Dr. Douglas A. Thom of Boston, Mass., spoke on the "Organization and Development of Mental Clinics for Community Care." The subject was discussed by Dr. A. Warren Stearns and Dr. E. Stanley Abbott. A second paper "A Community Mental Hygiene Clinic," was presented by Dr. Samuel Tartakoff of Taunton, Mass., and consisted mainly of a preliminary report of the work done at the Mental Hygiene Clinic in New Bedford, organized in April of this year and conducted by the Taunton State Hospital. It was discussed by Dr. George K. Pratt and Dr. W. B. Swift.

UNITED STATES PUBLIC HEALTH SERVICE

INTERCHANGE OF HEALTH OFFICERS IN THE UNITED STATES

An event which may be fraught with far-reaching consequences for world health is the Third General Interchange of Health Officers arranged by the Health Section of the League of Nations which is now taking place in the United States.

Representatives from France, England, Italy, Russia, Poland, Spain, Holland, Belgium, Greece, Jugoslavia, Germany, Switzerland, Norway, Mexico, San Salvador, Brazil, Chile, and Canada, delegated by their respective governments to participate in a course of study and observation, arrived in America the first week in September and will remain for approximately three months. Until September 22 the delegates will remain in Washington, studying national health organization as administered by the United States Public Health Service. Following the close of the course of study of the national health agency the delegation of visitors will separate into three groups, one of which will proceed to Virginia, another to North Carolina, and a third to Alabama, where about three weeks will be spent in studying state and local health departments. Richmond, Virginia, Raleigh, North Carolina and Montgomery, Alabama, are the Southern cities which have been selected for study. From there the groups will proceed to Massachusetts, New York and Pennsylvania, respectively, for the purpose of studying the health administration in three Northern States and small cities. Syracuse, New York, Allentown, Pennsylvania, and one of the smallest cities of Massachusetts, yet to be designated, will be visited for a short period. Following this the groups will make a study of health administration in three of the large cities of the East, namely, Boston, New York and Philadelphia. Present plans call for a reassembling of the entire delegation in Washington, late in November, for a final conference.

The present group numbers among its members many of the most eminent sanitarians of the world. France is represented by Dr. L. Aublant, Inspecteur principal des Services d'Hygiene, Dep. de l'Herault, and Dr. F. Bussiere, Directeur des Services d'Hygiene, de Montlucon. England is represented by Dr. Thos. Carnwath, D.S.O.M.D., Ministry of Health, London S. W. I., and Dr. Chas. Porter, M.D.M.O.H., St. Marylebone, London. Italy is

represented by Dr. F. Piccinni, Ufficio sanitario de Porto, Napoli. Russia is represented by Dr. S. Slonewski, San. Epid. Bureau, (2 Malyj-Tcherkassky Perenlok), Narkowzdraw, Moscou. Dr. A. Marzeew, Chef de San. Epid. (Lechnologiczskaja Ulica), Narkowzdraw, Kharkof, Ukraine, and M. Voeykoff, Sanitary Engineer, Narkowzdraw, Moscou. Spain is represented by Dr. R. Fernandez Cid, Inspecteur provincial de la Sante publique, San Sebastian. Holland is represented by Dr. D. J. Hulshoff Pol, Inspecteur gouvernemental de la Sante Publique, La Haye. Belgium is represented by Dr. van Boeckel, Directeur du Laboratoire de l'administration de l'Hygiene, Bruxelles, and Dr. Ensch, Chef de Service d'Hygiene, Schaerbeek, Bruxelles. Greece is represented by Dr. Prigos, Director, State Bacteriological Laboratory, Athens (a. b. s. Greek Legation, Paris). Poland is represented by Dr. J. Batko, Provincial Health Office (Wojewodski Uv ead Zdrowra), Cracow. Jugoslavia is represented by Dr. Ivo Kuhn, Referent du Ministere de la Sante publique, Belgrade. Germany is represented by Dr. K. Sannemann, Port Medical Officer, Hamburg. Switzerland is represented by Dr. J. Hunziker, Chef du Service d'Hygiene du Canton de Bale-Ville. Norway is represented by Dr. E. Anderson, Medicin de Prefecture, Lillehammer, Norvege. Mexico is represented by Dr. Enrique Orvananos, Government Inspector of Hygiene, Mexico City. San Salvador is represented by Dr. J. Segovea, Director de Sanidad San de Salvador. Brazil is represented by Dr. Vasconcellas. Chile is represented by Dr. J. Ducci, of the Faculty of Medicine, Santiago de Chile, and Dr. C. Mayers, Director, League of Social Hygiene, Santiago. Canada is represented by Dr. M. M. Seymour, Med. Deputy Minister of Public Health, and Director of Venereal Disease Control, Saskatchewan.

The delegation is accompanied by Dr. Norman V. Lothian of the Health Section of the League of Nations. Surgeon General Hugh S. Cumming, of the United States Public Health Service, delivered the address of welcome to the delegates.

THE YARDSTICK OF SUCCESS

INVESTIGATIVE work in medicine and the publication of the results thereof have come to be the yardstick by which the success of the individual and the excellence of the institution in which he works is measured. Medicine must progress or retrogress, for nature is a flux and there is no standstill, and that which does not go forward is left behind; it is one of the laws that bind us. Nevertheless, there are certain humanitarian laws which are imposed upon us as well, and a consideration of these should give us pause before we plunge ourselves too completely into an absorbed contemplation of science *in vitro*.

The unfortunate ones whose wounds must be dressed still demand an obligation of us; skill in the care of the sick must still be employed and should still be taught. It is worthy of comment that of a recent graduating class of a famous medical school only seventeen men were destined to enter general practice. The dearth of physicians able and willing to practice medicine in our rural communities is justly a matter of some concern. Is medicine and should medicine be a self-perpetuating science working in a general way only for the ultimate good of humanity in the abstract, or should one of its functions be to impart a little warmth and comfort to the sufferers at its door? It might be said that much flour is being milled but few are being taught to make bread.

The JOURNAL has long been of the opinion, and has before this expressed it, that the scientific achievements of the members of our medical faculties is worthy of great recognition, and it is glad that most of our medical schools have such individuals on their faculties. It is, however, also of the opinion that those rare men who, while capable of appreciating the advances of science have also a knowledge of the roots and branches of disease, and an understanding of their fellow men in sickness and in health and the quality of imparting some of their knowledge and of inspiring others to seek it, are worthy of still greater recognition, for they are rare indeed and their value is not to be taken lightly.

MEN OR ANIMALS?

THE current issue of *Hygiea* publishes an article by Ernest Harold Bayne, the naturalist, entitled, "Which Shall Live—Men or Animals?" This is another of the keenly pointed darts that Mr. Bayne, a deep student of the subject, has directed against anti-vivisection.

The conquest of bubonic plague, that killed 1,040,429 in India alone in 1905, is one example of the benefits that mankind has gained by animal experimentation, for by a series of experiments on rats, guinea-pigs and monkeys, the causative organism of the disease and the method of transmission were discovered, and as a result of these discoveries the knowledge of control has come.

The knowledge gained by the bedside observations of the pioneers in medicine is given its due credit, but the limitations of this method were absolute until experimentation upon living mammals led the way to newer and greater discoveries of inestimable value to mankind. The development of modern surgery is due in great part to vivisection. Antitetanic serum and antitoxin—in fact all the preventive and curative

serums—are the results of experiments on animals and are indeed themselves animal products. All our vaccines have been worked out and tested on mammals. It is through the use of animals that the benefits and dangers of drugs are tested.

Banting, by a series of experiments on dogs, completed the work on pancreatic function begun seventy-five years ago on rabbits by Claude Bernard, the friend and adviser of Pasteur, and gave insulin to mankind—ranking with salvarsan as one of the most brilliant medical discoveries of the century.

"Yet," Mr. Bayne continues, "the antivivisectionists boldly contradict all competent authorities and tell us that experiments on animals are useless, that they have never accomplished anything. The antivivisection societies are composed largely of well disposed but woefully misinformed persons. And those who are responsible for the misinformation are the leaders of the antivivisectionists. I have been studying these leaders for some years, and I may say, without any danger of my statements being disproved, that among them may be found many of the most dangerous of the criminal insane to be found in this country today."

After the presentation of this paper before the American Society of Mammalogists, the Society passed resolutions expressing itself as of the opinion that, "in the best interests of real humanity, animal experimentation, including vivisection, as practiced in our laboratories today, should continue unhampered."

The war against the antivivisectionists the antivaccinationists, and the medical liberty leagues must continue unremittingly, not in any hope of persuading them to see their errors, but to protect the mind of the public from their contamination. In doing this we must bear constantly in mind that their opposition is organized and that a bad cause, well organized, may win its victories.

THE MEDICAL SCHOOL DORMITORY

THE campaign for the Harvard Medical School dormitory is moving forward, but so far the response from the graduates themselves has not been what it should be. Physicians as a rule are not the wealthy members of the community. That a sum sufficient to build the proposed dormitory can be obtained from the graduates of the school is not to be expected nor is it intended. The dormitory will finally be erected through the offices of those public-spirited citizens who appreciate the services of our profession and are willing to invest their money in an enterprise, the returns from which will be in the form of improved service from that profession.

One guarantee will be demanded. Before this loyal support from the laity can be obtained our profession itself must prove by its own loyal support of the undertaking that it is a real and desired need. This can only be shown by a whole-hearted and unanimous response to the appeal that is being made for our money, and we must each respond according to our abilities, whether it be with one dollar or with one thousand.

The dormitory plan is primarily a humanitarian venture; "to give a proper and decent residence to those who would not otherwise have it," in the words of President Lowell to the dormitory committee. This object in itself, by improving the living conditions of the medical students, will add to the effectiveness of their later work and the usefulness of their profession.

Another, perhaps more tangible benefit to the community, will come from the solidarity of a large group of single-purposed men, living under the same roof and eating and playing and working together. This constant intermingling of young men with the same aspirations and ideals is of incalculable educational value, for, as President Lowell further said, "the spirit of a profession is one of the great moulding forces of the world."

CHICAGO'S SMOKE NUISANCE

THE Chicago Department of Health reports that the smoke nuisance in that city is caused by 300,000 apartment and residential buildings, 20,000 industrial plants and 1800 locomotives, and yet contends that the term "smoky city" need not apply, for measures can be instituted which will make the city clean. Smoke is spoken of as a damage to health, waste and damage to property and waste in fuel.

The record that in the time of Queen Elizabeth, 1580, a man was hanged for burning coal that smoked inordinately, is quoted. The concern of this Queen was aroused because of the fear that the health of her knights might suffer by having to breathe smoke and fumes.

The Chicago Department of Health expresses the opinion that smoke increases susceptibility to pneumonia because the presence of soot and small particles of coal irritate the linings of the air passages of the lungs, thereby preventing the exchange of pure gases and bringing about lowered resistance. In addition to these mechanical irritations, health is impaired by the reduction of the therapeutic effect of sunlight in a smoke-laden atmosphere, for cutting off sunlight lessens resistance to tuberculosis and the respiratory diseases. The record of Leeds is quoted to the effect that after six days of fog the infant mortality rate increased from 50 to 178 per thousand.

From an economic standpoint it is contended that the lowered efficiency of the people of Chi-

cago due to smoke imposes a great financial burden, and the waste and damage to property amounts to millions of dollars annually, and every individual in the city contributes to the $8 per capita annual cost. As an illustration, it costs the Chicago citizen $1.24 more for the washing of linen than the citizen of Philadelphia has to pay. The *Bulletin* then goes on to instruct the people how to care for fires in order to eliminate the production of smoke.

Even though this smoke nuisance may be greater in Chicago and Pittsburg than in many other cities, Boards of Health may properly engage in the study of these problems in their respective localities.

WORCESTER HEALTH REPORT

THE report of the Inspector of Milk in The Annual Report of the Board of Health of Worcester recognizes conditions which constitute a problem in many of our larger cities.

Out-of-State milk makes up nearly twenty-five per cent. of the supply and as the inspector states, there is absolutely no means of knowing under what conditions this milk is produced. The fact that it conforms to state and local standards as far as fat, bacteria count, etc., are concerned is not sufficient. The encouragement of a local supply is recommended.

Over seventy per cent. of the milk sold in the city, according to the report, is pasteurized, either properly or improperly. The compulsory installation of automatic temperature controls and temperature charts and the use of automatic bottling and capping machines is also recommended.

The inspector further warns against fancy but meaningless labelling of milk, and advises the public to consult with his office before selecting a milk supply.

Apparently Worcester is awake to the many-sided milk problem.

Miscellany

WAR ON "DOPE" PEDDLERS

A BITTER war is being waged between officials of the U. S. Veterans Bureau and members of "dope" gangs who are living off the war-earned compensation received by disabled ex-service men.

"I have instructed all employees of the U. S. Veterans Bureau to give every assistance to the narcotic forces of the Government in hunting out and running down these arch criminals who thrive on the blood and strength of their fellowmen," said General Frank T. Hines, Director of the U. S. Veterans Bureau, today in disclosing the recent results of the Bureau's war on dope peddlers and dealers.

The medical personnel of the U. S. Veterans Bureau, working in Veterans' hospitals, throughout the country, to restore health to the shattered bodies and minds of America's disabled former service men, have long realized that among their most deadly foes were those who sold pain-deadening but body-wrecking narcotics to the patients. There is probably no hospital in the country but has had its parasitic gang of dope peddlers and dealers.

It is an admitted fact among the medical profession that months of good medical treatment can be destroyed in a single day's contact by a patient with dope or liquor. To succeed in the battle for health necessitates the eradication of these outside damaging forces.

It is with this end in view that the Veterans Bureau officials, not themselves under any duty to act as assistants to the federal narcotic and prohibition forces, have undertaken to aid in the fight.

LET US BE FAIR

AFTER all, the emphasis that has been placed upon the early diagnosis and prompt treatment of diphtheria, and on the immediate use of antitoxin in those cases where diphtheria is to be thought of, the following conversation, which actually took place, is enlightening if not encouraging.

Speaking of a family in good circumstances and of unusual intelligence the remark was made, "They wouldn't have that doctor in the house again. Their baby had a sore throat and they called this doctor. He came and examined the child and without even taking a culture he gave it antitoxin and that night the baby had a high fever and was delirious. Why, they never heard of such a thing." . . . "Oh, yes, the baby got well."

Yet here was a doctor who treated that baby skillfully, conscientiously, and intelligently; who refused to endanger a baby's life by unnecessary delay in combating what might prove to be one of the most dreaded diseases of childhood.

Such logic as these parents used would have the fire department, at the sight of smoke and the sound of an alarm, send a fireman to investigate matters and then report back whether the apparatus should respond or not. One can readily imagine the result. In the same way, for that doctor to have waited a day for a culture report might have been just as disastrous to that child and more of a loss to those parents than an hour's delay in responding to an alarm of fire would be for some property owner.

Such criticism of any doctor is manifestly un-

just and harmful from whatever angle one views it. It is based entirely on prejudice or lack of knowledge. It is not much of an incentive to the doctor to render skillful, intelligent service if he knows that such service is not only unwelcome, but may actually serve to discredit him in the community.

The other side of the picture is this:

A bright, sturdy lad of five years complained of a sore throat. A doctor was called who left some medicine and went out. The next day the child was no better and the next day after that was decidedly worse. It was getting harder and harder for him to breathe. The same doctor was again called, again left some medicine and went out. Early the next morning the child had to fight for breath. Another doctor was called. This doctor saw that the boy had diphtheria and diphtheria in the worst form. He immediately gave the patient antitoxin, but to no avail. Within five hours the child lay dead—and all because no antitoxin had been given at the doctor's first visit.

Yet the doctor mentioned at the beginning of the story will be refused admittance into a certain house merely because he did his best, and succeeded, to prevent a tragedy like the one just described.—Bridgeport, Connecticut, Health Department Bulletin.

RED CROSS AND NATIONAL ASSOCIATION AGREE ON SEAL SALE

As the result of several conferences between representatives of the American Red Cross and the National Tuberculosis Association, the following general points of agreement have been reached, and are published for the information and guidance of all tuberculosis workers engaged in the next Christmas seal sale.

(1) That neither the Red Cross nor the Tuberculosis Association had military authority over their chapters or affiliated associations.

(2) It was agreed that the American Red Cross and the National Tuberculosis Association would endeavor in so far as was practicable to prevent unnecessary overlapping, especially in such instances as outlined above.

(3) It was agreed that the chapters and affiliated associations would again be advised that there was no official connection between the Christmas seal sale campaign of the National Tuberculosis Association and the Roll Call of the American Red Cross.

(4) It was agreed that for the year 1923 the American Red Cross Roll Call will be held from Armistice Day, November 11, until Thanksgiving Day, November 29, and that the Tuberculosis Christmas seal sale will not begin until November 29.

(5) It was agreed by the American Red Cross that they would again call to the attention of their chapters the importance of not carrying on their campaign after Thanksgiving Day and that it would endeavor to prevent the carrying out of a Roll Call during the month of December.

(6) The National Tuberculosis Association agreed that it would urge its affiliated associations to advise volunteer workers of the difference between the Red Cross Roll Call and the Tuberculosis Christmas seal campaign and that the seal should always be referred to as the Tuberculosis Christmas Seal.

(7) The National Tuberculosis Association also agreed that it would advise newspapers and photographers that the National Tuberculosis Association is engaged in selling a Tuberculosis Christmas seal and that this seal is not a Red Cross seal.—*Bulletin of the National Tuberculosis Association.*

NEAR EAST RELIEF

The Near East Relief, incorporated by an Act of Congress, is feeding approximately two hundred thousand children located in orphanages and other institutions in Armenia, Syria, the Caucasus, Greece and the Isles of the Aegean Sea. This is the largest child-feeding clinic in the history of the world.

The lives of these children have depended and still depend upon the generosity of the American public. To support these children, the Near East Relief makes various appeals to collect money, clothing and food.

Naturally, with so many dependent children on our hands, one of the greatest appeals must be for food, and milk especially.

It has been the intention of the N. E. R. to place the milk appeal before the public in such a way that each American man and woman would feel as if they were personally feeding a hungry child at their own home.

Condensed milk drives are being conducted in various cities for a week at a time, asking the people for donations in merchandise. Arrangements are made in each city for the proper amount of publicity through the newspapers—the railroads coöperating by hauling all donated milk free of charge to the seaport.

Condensed milk has been successfully used by the N. E. R. as an article of diet for infants and older children for four years, owing to the fact that war and pestilence have entirely eliminated all sources of local supply of milk.

J. C. Curran, M.D., Director of Medical Service, 151 Fifth Avenue, New York, will give any desired information.

BOARD OF REGISTRATION IN MEDICINE

September Examination, 1923

Examined	40
Registered	17
Rejected	21

One dismissed for cribbing—Graduate of St. Louis College Physicians and Surgeons.

One unable to speak and write English, therefore not eligible to remain in examination—Graduate University of Athens.

REGISTERED

St. Louis College Physicians and Surgeons	2
Chicago Homeopathic Medical College	1
Physicians and Surgeons, Boston	1
Middlesex College Medicine and Surgery	1
Massachusetts College Osteopathy	2
Tufts College Medical School	3
Chicago College Osteopathy	1
University of Naples	1
University of Medicine, Beirut	1
Harvard Medical School	3
Temple University	1
Total	17

REJECTED

St. Louis College Physicians and Surgeons	5
Physicians and Surgeons, Boston	3
Middlesex College Medicine and Surgery	5
Massachusetts College Osteopathy	2
Philadelphia College Osteopathy	1
University Naples	2
University of Geneva	1
University West Tennessee	1
Long Island College Hospital	1
Total	21

PHYSICIANS REGISTERED IN SEPTEMBER EXAMINATION

Bloomberg, John Rubin, 199 Harvard Street, Malden, Mass.

Brusch, Joseph Armao, 88 Francis Street, Boston, Mass.

Butler, William Herbert, 27 Dana Street, Cambridge, Mass.

Chiaramida, Joseph, General Delivery, Lawrence, Mass.

Creed, John Patrick, 31 Union Street, Haverhill, Mass.

Federici, Louis Mario Nicomede, 453 Central Avenue, Orange, New Jersey.

Gleason, Joseph Henry, 60 Court Street, New Bedford, Mass.

Hagopian, Michael, 17 Kenneson Road, Somerville, Mass.

Jones, Wilger Lancaster, 8 Boyden Street, Worcester, Mass.

Keeley, Thomas Henry, Lynn Hospital, Lynn, Mass.

Merlin, Samuel Abraham, 43 Compton Street, Boston, Mass.

Powers, John Paul, Worcester State Hospital, 166 Summer Street, Worcester, Mass.

Scheller, Louis, 25 Carver Street, Cambridge, Mass.

Smith, Edward Charles, Boston City Hospital, Boston, Mass.

Stanton, Frank Daniel, 551 Eliott Street, Milton, Mass.

Staro, Humbert, 221 East 121st Street, New York City, N. Y.

Woodbridge, Philip Dudley, 745 Massachusetts Avenue, Boston, Mass.

SUSPENDED REGISTRATIONS

At a meeting of the Massachusetts Board of Registration in Medicine held October 4, 1923, the certificate issued to Earl R. Devere of Millville was suspended for one month on complaint of a son of a woman who had died of cancer. It was alleged that the woman had been treated at the Huntington Hospital but was induced to secure the services of Dr. Devere. She was led to expect benefit from the treatment, which consisted in the injection of a fluid and the use of pills. The treatment was continued from November, 1922, to April, 1923. Four hundred and seventy-five dollars was paid for the treatments. The Board construed the attitude and behavior of Dr. Devere as unprofessional conduct as specified in the law.

The certificate of Dr. Fred M. McKenney of Boston was suspended for one month for violation of the prohibition laws.

CANCER RESEARCH

An experiment that may explain the essential nature of cancer has been announced by the Imperial Cancer Research Fund in London, according to information reaching the American Medical Association at Chicago. It was found that cells from cancerous growths will grow outside the body if they are planted in a suitable medium, but that ordinary body cells such as those in the kidney will grow only after they have been removed from the body for several weeks. But the ordinary cells were found to grow immediately if some of the extract in which a tumor is growing is added to the ordinary well cells. The British scientists argued from this that cancer cells automatically and continuously produce substances which activate growth and that normal cells produce these substances only when injured.

Long continued irritation of tissues may produce cancer, past experience has shown; and the findings of the British scientists explain that this is in accord with the fact. Vitamin deficiency has nothing to do with the growth of cancer the announcement also stated on the strength of extensive researches.—*Science.*

DIRECTORY OF NEUROLOGISTS AND PSYCHIATRISTS

THE National Committee for Mental Hygiene is compiling a directory of all physicians in the country who are practicing neurology or psychiatry, with autobiographical information concerning each, so far as possible. It will bring to date the neuropsychiatric resources now available. This information will be used to meet the demand for the services of such specialists in different communities, as well as the increased demand for the services of such specialists in positions.—*Mental Hygiene Bulletin.*

THE NEW PRESIDENT OF THE BRITISH ASSOCIATION

MAJOR-GENERAL SIR DAVID BRUCE, F.R.S., has been honored by election to the presidency of this Association. The next meeting of this body will be held in Toronto in 1924.

Sir David is credited with having discovered the Micrococcus Melitemsis, which is accepted as the cause of Malta fever. He has added much to the knowledge of the tsetse fly disease and other insect-borne diseases. He is also credited with having been an important factor in the discovery of the cause and treatment of trench fever.

DIABETIC COLUMN

ALCOHOL AND DIABETES

THE prescription of alcohol for diabetes gained favor, in America at least, following Allen's[1] statement in 1914. In describing his method of ridding diabetics of glycosuria and acidosis, he said, "Plain fasting suffices for the purpose, but since alcohol is a food which does not produce glycosuria and *is said*[2] to diminish ketonuria, it is generally given during fasting, especially if there is danger of coma." During the following year "A Study of Acidosis in Three Normal Subjects with Incidental Observations on the Action of Alcohol as an Antiketogenic Agent," was made by H. L. Higgins, F. W. Peabody and R. Fitz.[3] They found that "Alcohol given to the subjects on this diet in dosage comparable to that used for clinical purposes did not stop the progress of the acidosis or show any antiketogenic action. Coincidental with its demonstration there was further increase in the oxygen consumption and in the disagreeable subjective symptoms." Joslin[4] in 1916 wrote about alcohol as follows: "It has been used by me less and less of late and at no time have I prescribed as much as 30 c.c. every three hours. One should not prescribe alcohol thoughtlessly for diabetics."

Since 1916 the larger diabetic clinics in this country have used very little alcohol. In deference, however, to its common use among practitioners, the question was brought up a few days ago before a meeting of fifteen Boston men interested in the treatment of diabetes, representing the clinics of the New England Deaconess Hospital, Massachusetts General Hospital, Peter Bent Brigham Hospital, Boston City Hospital. Only four had used alcohol in any form in the past four years. None had given it to more than two patients in this time. In no case had it been given for the diabetes. In three cases it was given as a concession to a habit, and in the other two in order to administer a bad tasting preparation of fat. In other words, for only five out of at least 3000 cases treated by members of this group had alcohol been prescribed.

It is not fair in this connection to omit the statement that so experienced a diabetic student as Van Noorden[5] advocated the use of alcohol in diabetes as late as December 13, 1922, in an address before a Berlin Medical Society.

Some of the reasons for not using alcohol are:

1. It may be very difficult to distinguish the coma of acidosis from the unconsciousness following too much Insulin. If alcoholic stupor were a third possibility the problem might be even greater.

2. Anuria is occasionally a cause of death in diabetes. Several of the cases that have died of anuria have been given nothing but alcohol for varying periods of time prior to the onset of the anuria.

3. A great many fasting diabetics when given alcohol objected to it because it irritated their stomachs and did not in any way allay the disagreeable features of the fast.

4. Experience teaches that alcohol is unnecessary.

5. Generalized neuritis in diabetes, according to Labbé,[6] is most often due to alcohol or to alcohol plus diabetes.

6. At least twice when patients have of their own volition taken alcohol following the injection of Insulin, severe Insulin reactions have occurred.

ALBERT A. HORNOR, M.D.

1 The Treatment of Diabetes: F. M. Allen, M.D., N. Y. BOSTON MED. AND SURG. JOUR., 1915, 172, p. 241.
2 Neubauer, Otto: Münch. med. Wochenschr., 1916, 791.
3 Jour. Med. Res., 1916, 29, p. 263.
4 Treatment of Diabetes Mellitus (1st edition), Lea and Febiger, 1916, p. 227.
5 Van Noorden, C.: Med. Klin., Berlin, 1923, 19, p. 41.
6 Labbé, Marcel, (Paris). A Clinical Treatise on Diabetes Mellitus, translated by Cumston, Charles G. Wm. Wood & Co. N. Y., 1922.

News Items

NOTES ON TUFTS COLLEGE MEDICAL SCHOOL.—Tufts College Medical School opened this autumn with a registration of 155 students in the first-year class, the largest class that has been admitted to the school in recent years. There were 300 applicants from whom the successful candidates were selected. The total registration in the school is 515.

The intelligence tests which were introduced last year will be continued each year on the incoming class until sufficient data have been compiled to make a statistical study of some value. Although great interest is manifested in the subject of intelligence tests, it is by no means clear what the value of these tests is, and whether they are applicable in a professional school as in a school which aims to give a general education.

A system of faculty advisers to students has been introduced, but the details of the system will be worked out from time to time as the year progresses.

Through discussion in the department of Neurology an effort will be made to emphasize the point of view that the patient is a person who is sick, not merely a case of disease. Psychology is now one of the required subjects in preparation for work in medicine, but it is rather general psychology than the psychology which is of greatest value to the physician in understanding the social maladjustment of his patients. The group of concepts contained in social psychology is perhaps more useful to the physician who has taken a course in general psychology.

Dr. Frank H. Lahey, who has been appointed Professor of Surgery at Harvard, will continue as head of the department of surgery at Tufts College Medical School.

Among the changes on the faculty will be noted the appointment of Dr. Martin J. English, Assistant Professor of Pediatrics, and Dr. George Sykes, Assistant Professor of Anatomy.

REMOVALS.—Dr. James F. McLaughlin has moved from the Springfield Hospital to 25 Park Street, Adams, Mass.

Dr. Arthur E. Brides has moved from Cambridge to 167 Hicks Street, Brooklyn, New York.

Dr. William Reid Morrison has moved his office from 527 Beacon street, to 520 Commonwealth Avenue, Boston.

Dr. Emily A. Pratt is now at 33 Central Avenue, St. George, Staten Island, N. Y.

Dr. O. R. Fountain has moved from Lynn to Rowley.

Dr. H. A. Novack has moved from Dorchester to 373 Harvard Street, Brookline.

Dr. Edward A. Pease, for some time at Westbrook, Conn., is now at the St. Botolph Club, Boston.

Dr. H. A. Schneider has moved from Pittsfield to the State Colony at East Gardner.

Dr. Charles Orrin Day has removed his office to 298 Marlborough Street, Boston, Mass.

SYNTHETIC INSULIN.—*The Medical Press and Circular* states that the reports of the use of synthetic insulin are encouraging, and if found equal to the effect of the product made from the pancreas, will reduce the cost to a considerable degree.

GLAND TRANSPLANTATION.—Dr. S. Veronoff spoke before the Second International Congress of Comparative Pathology at the meeting in Rome October 7. He related the results of his operations on forty-five men and described his technique.

ANNOUNCEMENT.—Dr. Frank K. Hallock announces that he has associated with him in carrying on the work of Cromwell Hall, Ray Lester Whitney, Ph.B., M.D., a graduate of Brown University and of the Harvard Medical School.

DR. FLEXNER HONORED.—The French Government took advantage of the Pasteur Centenary to confer upon Dr. Simon Flexner, director of the Rockefeller Institute for Medical Research, the rank of Commander of the Legion of Honor.

BIOGRAPHY OF GENERAL GORGAS.—A biography of the late Surgeon-General William C. Gorgas, now in preparation by his widow in collaboration with Burton J. Hendrich, is soon to be published.

DR. RICHARD P. BLACK of Peak's Island, who was recently arrested and charged with murder and abortion, as a result of the death of one Mrs. Ilene York, was found not guilty, after due trial, and acquitted in the Superior Court, Portland.

ANNOUNCEMENT.—Dr. Benedict Fenwick Boland, who resigned as Resident Surgeon of the Boston City Hospital, October 1st, announces the opening of his office at 270 Commonwealth Avenue.

WEEK'S DEATH RATE IN BOSTON.—During the week ending October 6, 1923, the number of deaths reported was 200, against 208 last year, with a rate of 13.54. There were 22 deaths under one year of age, against 30 last year.

The number of cases of principal reportable diseases were: Diphtheria, 46; scarlet fever, 35; measles, 9; whooping cough, 19; typhoid fever, 10; tuberculosis, 45.

Included in the above, were the following cases of non-residents: Diphtheria, 9; scarlet fever, 4; typhoid fever, 1; tuberculosis, 4.

Total deaths from these diseases were: Diphtheria, 4; tuberculosis, 8.

Included in the above, were the following cases of non-residents: Diphtheria, 3; tuberculosis, 2.

Correspondence

THE PRUDENTIAL INSURANCE COMPANY OF AMERICA

Home Office, Newark, New Jersey

October 10, 1923.

Dr. W. P. Bowers, Managing Editor,
Boston Medical and Surgical Journal,
126 Massachusetts Avenue, Boston, Mass.

My dear Sir:

You may wish to give space to the following:

The National Belgian League for the Control of Cancer expects to hold a National Congress, the sessions of which will take place in Brussels on November 18, 19 and 20. The opening session will be addressed by Viscount Paul Berryer, Minister of the Interior and Hygiene, followed by an address on "The Organization and Initial Results of the Fight against Cancer in France," by Professor Bergonne of Bordeaux.

Among other speakers will be Professor Hartmann of Paris, on "The Early Diagnosis of Cancer"; Professor Depage, who is well known in this country, on "The Influence of Surgery in the Fight against Cancer"; Professor Firket of Liege, on "The Diagnostic Microscopic Service in the Province of Liege"; Professor Goris of Brussels, Professor Mayer of Brussels, and others.

The American Society for the Control of Cancer will be represented by Dr. F. L. Hoffman, consulting statistician of the Prudential Insurance Company of America, who will deliver an address on "Cancer and Civilization."

The sessions will be presided over by the Belgian Queen.

Very truly yours,
F. L. Hoffman, *Consulting Statistician.*

ACKNOWLEDGMENT OF BOOKS FOR REVIEW

The Journal acknowledges the receipt of the following books for review:

Diagnostic Methods. Webster. P. Blakiston's Son & Co. Pp. 909. Cloth, $8.

Chronologia Medica. Sir D'Arcy Power. Paul B. Hoeber. Pp. 278. Price $3.50.

Manual on Ship Sanitation and First-Aid for Merchant Seamen. Second edition. Robert W. Hart. New York: Seamen's Church Institute of New York. Pp. 188. Price $1.

Practical Dietetics—Diet in Health and Disease. Alida Frances Pattee. Mt. Vernon, N. Y.: A. F. Pattee. Pp. 687. Price $2.60.

Aids to Gynaecology. Richard E. Tottenham. Sixth edition. New York: William Wood & Co. Pp. 128.

Asthma. Frank Coke. New York: William Wood & Co. Pp. 260.

Papers from the Mayo Foundation and the Medical School of the University of Minnesota. II. 1921-22. Philadelphia and London: W. B. Saunders Co. Pp. 716. Price $10.

Excursions into Surgical Subjects. John B. Deaver and Stanley R. Reimann. Philadelphia and London: W. B. Saunders Co. Pp. 188. Price $4.50.

Heart Records: Their Interpretation and Preparation. S. Calvin Smith. Philadelphia: F. A. Davis Co. Pp. 313. Price $7.

Traite d'Ophthalmologie. Poulard. Two volumes. Paris: Masson et Cie. Pp. 1457. Price 120 fr.

Endocrine Diseases—Including Their Diagnosis and Treatment. Wilhelm Falta. Third edition. Philadelphia: P. Blakiston's Son & Co. Pp. 669. Price $8.50.

A Practical Handbook of Diseases of the Ear. William Milligan and Wyatt Wingrave. New York: William Wood & Co. Pp. 191. Price $3.50.

The Ophthalmic Year-Book. Vol. xix, 1923. Ophthalmic Publishing Co., Chicago. Pp. 390.

The Practical Medicine Series. Vol. I, General Medicine. Series, 1923. Year Book Publishers. Pp. 678.

Fuss und Bein. Dr. Georg Hohmann. Munchen: J. F. Bergmann. Pp. 182, 17 tables. Gz. 10.50.

Handbook for Mental Nurses. Seventh edition. Chicago: Chicago Medical Book Co. Pp. 615. Price $2.50.

DEATH NOTICE

Dr. Charles Moran Atchison, formerly of New Bedford, a fellow of the Massachusetts Medical Society, died at the naval hospital, Vallejo, California, September 27, 1923, of gastro-enteritis, at the age of forty-one. He was a graduate of Tufts College Medical School in 1908, and had practised in New Bedford until he entered the Medical Corps of the Navy. Serving first in the Medical Corps of the Massachusetts Militia, he was in the transport service during the war and entered the Medical Corps of the Navy in 1921. Later he had been stationed at the Mare Island Naval Station Hospital, San Francisco, with the rank of lieutenant. He is survived by his widow, who was Miss Hannah E. Norton, of New Bedford, and by one daughter.

NOTICES

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

Meetings of this society will be held at Norfolk County Hospital, Braintree, Mass., the first Thursday of each month until further notice.

HAMPSHIRE DISTRICT MEDICAL SOCIETY

The next meeting of the Hampshire District Medical Society will be held in Northampton, November 14, 1923, at 4.30 p. m. Dr. Howard F. Root of Boston will read a paper on the "Insulin Treatment of Diabetes."

Regular meetings are held bi-monthly, the second Wednesday in the month, at Boyden's Restaurant, Northampton.

E. E. Thomas, M.D., *Secretary.*

AUSTRALASIAN MEDICAL CONGRESS

The Australasian Medical Congress (British Medical Association) meets in Melbourne, Victoria, November 13 to 19.

CENSORS' MEETING

The Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, November 1, 1923, at 4 o'clock.

Candidates should make personal application to the Secretary, and present their medical diploma at least one week before the examination.

LESLEY H. SPOONER, *Secretary.*
520 Commonwealth Avenue, Boston, Mass.

UNITED STATES CIVIL SERVICE EXAMINATION

The United States Civil Service Commission announces open competitive examinations for the positions of Inspector and Agent, Antinarcotic Act. The entrance salaries for both agents and inspectors will range from $1800 to $2250 a year plus bonus, subsistence allowance and necessary travelling expenses.

Applications must be filed with the Civil Service Commission, Washington, D. C., by November 6, 1923.

Physicians and pharmacists may be selected as inspectors.

BUSINESS MEETING OF THE NEW ENGLAND SURGICAL SOCIETY

The business meeting, notice of which was omitted from the program, will be held Thursday, October 18, at 2 p.m.

THE CUTTER LECTURE ON PREVENTIVE MEDICINE

BY SIR ARNOLD THEILER, K.C.M.G.

Director of Veterinary Education and Research, Union of South Africa; Professor of Animal Pathology, University of South Africa, etc.

Phosphorus Deficiency in Animals: Its Effects and Prevention. Wednesday, October 17, 1923, at the Harvard Medical School, Amphitheatre Building E, at 5 p. m.

These lectures are given annually under the terms of a bequest from John Clarence Cutter, whose will provided that the lectures so given should be styled the Cutter Lectures on Preventive Medicine and that they should be delivered in Boston and be free to the medical profession and the press

DISEASES REPORTED TO MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

WEEK ENDING OCTOBER 6, 1923

Disease	*No. of Cases*	*Disease*	*No. of Cases*
Anterior poliomyelitis	12	Pneumonia, lobar	39
Chicken-pox	96	Scarlet fever	119
Diphtheria	191	Septic sore throat	2
Dog-bite requiring anti-rabic treatment	1	Suppurative conjunctivitis	12
Epidemic cerebrospinal meningitis	3	Syphilis	47
German measles	4	Tuberculosis, pulmonary	127
Gonorrhea	96	Tuberculosis, other forms	13
Influenza	4	Typhoid fever	27
Malaria	1	Whooping-cough	78
Measles	116	Tetanus	1
Mumps	44		
Ophthalmia neonatorum	11		

SOCIETY MEETINGS

DISTRICT SOCIETIES

Suffolk District Medical Society:

October 31, 1923:—Stated Meeting at the Boston Medical Library at 8.15 p. m.

November 14, 1923:—Meeting of Surgical Section in association with the Middlesex South District Medical Society at the Boston Medical Library at 8.15 p. m.

December 19, 1923:—Meeting of Medical Section at the Boston Medical Library at 8.15 p. m.

January 30, 1924:—In association with the Boston Medical Library and the Middlesex South District Medical Society at the Boston Medical Library at 8.15 p. m.

February 27, 1924:—Meeting of Surgical Section, in association with the Middlesex South District at the Boston Medical Library at 8.15 p. m.

March 26, 1924:—Meeting of the Medical Section, in association with the Boston Association for the Prevention and Relief of Heart Disease, at the Boston Medical Library at 8.15 p. m.

April 30, 1924:—Annual Meeting, to be held at the Boston Medical Library at 8.15 p. m.

Worcester District:—The meetings for the year are as follows:

November 14 in Whitinsville. Paper by Dr. Channing Frothingham of Boston.

December 12 in Worcester. Papers by Dr. Fred B. Lund of Boston and Dr. Michael F. Fallon and Dr. Walter Seelye of Worcester.

January 9 at St. Vincent Hospital, Worcester.
February 13 at Memorial Hospital, Worcester.
March 13 at City Hospital, Worcester.
April 10—A public meeting.
May 8—Annual Meeting.

Franklin District:—Society meets at Greenfield the second Tuesday of November, January, March, May, July, September. Annual Meeting in May.

Norfolk South District:—Meetings first Thursday of each month at 11.30 a. m. October at Norfolk County Hospital, Braintree; November, December, January, February, March, April, and May at United States Hotel, Boston. The November, February and May meetings are Stated Meetings.

Essex North:—Combined meeting with Middlesex North, Middlesex East and Essex South October 17, at 1.30 p. m., at North Reading State Sanatorium, North Reading. Semi-annual meeting at Haverhill, January 2, 1924. Annual Meeting at Lawrence, May 7, 1924.

Middlesex North:—Combined meeting with Middlesex East and Essex South, October 17, at North Reading Sanatorium. January 31, 1924, at Lowell. Annual Meeting in April.

Middlesex South District Medical Society:

November 14, 1923:—Combined meeting with the Surgical Section of the Suffolk District Medical Society at the Boston Medical Library.

December, 1923:—Hospital Day, probably at the Cambridge Hospital.

January 30, 1924:—Combined meeting with Suffolk District at the Boston Medical Library.

February 27, 1924:—Combined meeting with the Surgical Section of Suffolk District at the Boston Medical Library.

March, 1924:—Hospital meeting; place not yet determined.

April, 1924:—Annual meeting.

Hampden District:—The meetings for the year are as follows:

Combined meeting October 26, 1923, at Hotel Kimball, Springfield, at 1 p. m. Speakers: Dr. J. S. Stone of Boston, Dr. Alfred Worcester of Waltham, Dr. E. H. Bigelow of Framingham, and Dr. W. A Pusey of Chicago. January, 1924, at Springfield. April, 1924, at Springfield. Annual Meeting.

Bristol South—Semi-annual meeting will be held in New Bedford, November 1, 1923. The Annual Meeting will be held in New Bedford, May 1, 1924.

STATE, INTERSTATE AND NATIONAL SOCIETIES

October 17-18, 1923:—Annual Meeting of New England Surgical Society in Boston.

Schedule of meetings of the New England Dermatological Society:

Monday, November 5, 1923, at 2 p. m., in the Skin Out-Patient Department, Massachusetts General Hospital.

Wednesday, December 12, 1923, at 3 p. m., in the Surgical Amphitheatre, Boston City Hospital.

Wednesday, February 13, 1924, at 3 p. m., in the Skin Out-Patient Department, Massachusetts General Hospital.

Wednesday, April 9, 1924, at 3 p. m., in the Surgical Amphitheatre, Boston City Hospital.

Eastern Dermatological Societies:—The second annual joint meeting of these societies will be held at the Massachusetts General Hospital, in the Skin Out-Patient Department, on November 5, 1923, at 2 o'clock.

For list of Officers of the Massachusetts Medical Society, see page x of the Advertising Section.